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SYMPOSIUM

THE NERVUS ACUSTICUS.

I.—APPLIED ANATOMY OF THE VIIIth NERVE AND ITS ENVIRONS, THE CEREBELLOPONTILE ANGLE.*†

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In recent years, attention has been more or less diverted from the nervous connections of the ear by investigative work of more purely clinical import dealing with diseases of the middle ear and mastoid. An almost single redeeming feature to this situation has been the introduction of the science of neuro-otology, which has again called attention to the fact that intracranial disorders may superficially simulate disease within the petrous bone. This series of studies on the nervus acusticus has been planned to restore, in part at least, the proper balance of attention between the middle ear and end organs on the one hand and the nervus acusticus on the other. We are to explore a field of common importance to the otologist, the neurologist and the neurosurgeon. A proper evaluation of the symptoms and signs provoked by lesions of the eighth nerve can be had only by a knowledge of its relationships and the diseases which affect it. This is certainly so in detecting certain intracranial extensions of infec-

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tious processes having their origin in the middle ear and mastoid which involve primarily or secondarily the vestibular pathways.

It is not the purpose of this introductory paper to rewrite the anatomy of the eighth nerve or its associated structures and nervous connections, but rather to correlate the essential features which have a direct bearing on the production of symptoms in the course of its diseases. A detailed description of the end organs is likewise out of order and will not be considered. While the nervus acusticus and its environs have been especially restudied *in situ*, much of the material here presented is necessarily of the nature of a review. A prefatory historical note will serve as a third dimension to matters more purely anatomical.

Matters Historical: The nervus acusticus, like many other anatomic entities, has had an interesting history. The first curious investigator to visualize this tiny nerve and to propose a function for it has long since been forgotten. In his memorable volumes on anatomy, Vesalius[1] (1725) failed to utilize his usual care in observation in his description of the cranial nerves. His illustrations of the base of the brain and the cerebellopontile angle make it difficult to determine just which of his "seven pair" are the auditory nerves. Included with the nervus facialis, it was either the fourth or more likely the fifth pair (see Fig. 1). It is likely that he followed a current conception of his day as echoed by contemporaries and followers who considered the two nerves collectively as the *fifth* cranial nerve (Veslingus,[2] 1659).

Winslow[3] (1749) described ten pair of nerves, the tenth consisting of the first cervical roots (n. suboccipitalis). The seventh pair he designated as the n. auditorii, indicating its two obvious divisions as the *portio mollis* (n. acusticus) and the *portio dura* (n. facialis), which terms persisted well into the nineteenth century. Cheselden[4] (1750) in describing this nerve stated that—

"The seventh pair coming out from the side of the root of the annular process and entering the meatus auditorius internus, and immediately dividing, one part soon loses its firm coats, and is expanded into the innermost camera of the ear" . . .

Soemmerring[5] (1794-1801) was one of the first anatomists to recognize twelve individual nerves, although the subdivisions of his predecessors renders this distinction somewhat superficial.

Sir Charles Bell[6]* (1811), the father of modern neurology, followed the older classification of the nerves, but described quite

*On pages 440 and 441 of Bell's treatise is found a condensed history of the classification of the cranial nerves. That dealing with the "seventh pair" is here appended.

accurately the anatomic structure and course of the nervus acusticus. Spurzheim[7] (1834) emphasizes its comparative anatomy as well as its relationship to the striae medullares acusticae. In his steel engravings, Tiedemann[8] (1826) illustrated the occurrence of the eighth nerve in a 27-week-old fetus. As late as 1849, the American anatomist, Morten[9], described the cranial nerves as nine pair, in spite of the increasing tendency to name twelve anatomically separate ones.

7th pair
- Auditory nerves
  - 2um par Alexand. Benedict.
  - 4um par Carol. Stephan.
  - 5um par Vesal. et aliorum.
  - 6um par V. Horne
  - Portia mollis, of the Moderns.
- Nervus communicans faciei
  - Distinctus a molli nervus Fallop.
  - Portio ut praecedens 5 Paris, ed.
  - est 7 recentiorum Vesal.
  - Portio dura, of the Moderns.
  - Le petit sympathique, of Winslow
  - Facial nerve.

COMPARATIVE ANATOMY.

The study of the VIII nerve in animals, particularly of representative vertebrates, brings to light two important and interesting aspects. With the exception of certain fish, as mentioned by Spurzheim, in which this nerve has been mistaken for the nervus trigeminus or nervus facialis, its origin is usually found at the lateral extremity of the juncture of the pons and medulla. This is explained largely by the fairly constant position of its end organ in relation to the skull and brain as a whole. In this respect there is a close analogy to the nervus acusticus and its end organs in the human. In the second place, the relative size of the nerve varies in proportion to the importance of its function in the individual animal concerned. It is relatively larger in the porpoise[10]. In the accompanying illustration (see Fig. 2) the anatomic relations of the nervus acusticus in various vertebrates is indicated. In addition to this close relationship between size and function, there exists the possibility that certain individual variations may also exist. Santorini[11] described a case of a blind man with unusually acute hearing, in whom a noticeably large auditory nerve was found at autopsy.

DEVELOPMENTAL ASPECTS.

In order to be at all comprehensive, an embryologic consideration must include three items: *1*. The development of the nerve and its ganglia; *2*. the development of its central connections, especially the structures in the recessus lateralis, and *3*. that of the bony structures which form the enveloping canal and meatus auditorius internus. To date we have no detailed information as to the development of the meninges covering the posterior surface of the petrous bone.

One can only conjecture, therefore, as to the significance of certain neoplastic lesions arising therefrom.

Development of the Nervus Acusticus: The development of this nerve presents a few most interesting and apparently fundamental facts as utilized by some in interpreting its pathology. In the human embryo, the first evidence of the eighth nerve and its ganglia is a mass of epithelial cells which has migrated from the environs of the encephalic tube to a point anterior to the auditory vesicle. This cell group, known at this stage as the ganglion acustico-facialis, subsequently migrates to a position medial to the vesicle to lie in the mesenchyme between it and the walls of the rhombencephalon. Very early

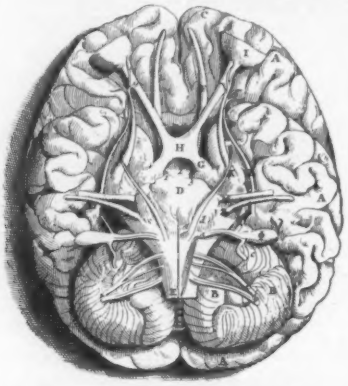

Fig. 1. Vesalius' drawing of the base of the brain. From its relationship to the abducens (?), the nerve root indicated as (a) probably includes the nervus acusticus.

in the process, the ganglion geniculatus separates itself from its dorsal aspect to become a distinct structure. By the time this has taken place, fibres from the cells in the ventral portion have reached the recessus lateralis and assume the proportions of a nerve. The ganglion is now further subdivided into a superior (vestibular) and an inferior (cochlear) portion (see Fig. 3). The constituent ganglion cells are bipolar in type and proliferate peripheral and central processes. The longer central fibres extend into the rhombencephalon where connections are made with groups of cells destined to become the vestibular and cochlear nuclei.

Of special interest in this connection is the fact that the fibres of the vestibular nerve cells grow faster and their entrance into the brain stem at the recessus lateralis actually precedes those of the

acoustic portion. The peripheral portions of both come to be covered with sheath cells, differentiated along with the neurons from a common parent cell. The process of this differentiation need not be considered in detail here. The fibres of the central portion of the nerve, to the contrary, are surrounded by neuroglia which have migrated into the nerve from the adjacent lateral recess. Inasmuch as the vestibular fibres reach the pons earlier, the glial cells migrate further into this portion of the nerve (see Fig. 4A). This embryologic fact has been utilized, first by Henschen[12] and more recently by Skinner[13], to explain the apparent excess of sheath cells in the vestibular portion of the ganglion. In other words, because the glial cells have an early start in this part of the nerve, they extend a greater distance into it. The sheath cells, finding that part of their work has already been taken care of, accumulate in an irregular fashion in the ganglion.

That this might be the case is further suggested by a difference in the arrangement of sheath cells in the vestibular ganglion as occurs in the older embryos. The sheath cells in the cochlear ganglion form well defined and regularly disposed capsules for both the nerve cells and their fibres, as is the case in the dorsal root ganglia (see Fig. 4C). To the contrary, the sheath cells in the vestibular ganglion have no well defined arrangement and lie to a large extent in a confused manner between the somewhat widely separated nerve cells (see Fig. 4D). It is this apparent excess of sheath cells in the vestibular portion of the ganglion and nerve that is thought to give rise to neurinomas common to the nervus acusticus. In support of this are the clinicopathological demonstrations of Henschen that this tumor usually has its origin in this situation. That this is not always the case is suggested in the study of Morelle[14]. The details of the development of the eighth nerve may be found in the contributions of His[15], Weigner[16] and Streeter[17].

Development of Central Connections and the Recessus Lateralis: As has already been stated, the vestibular portion of the nerve reaches the recessus lateralis prior to the auditory portion. In the 20-30 m.m. human embryos, the two parts of the nerve can be distinguished and can be seen entering the rhombencephalon as separate structures. Before this, however, due to the development of the pontine flexure, a transverse groove is formed across its floor. This results in a prominence or outpocketing on either side continuous with the narrow approach to the ventricle, the lateral recess. Cellular proliferation and migration takes place rapidly in the rhombic lip leading to the formation of the nuclei of reception. Coincident with the sepa-

ration of the two constituents of the nerve, the nuclear mass also divides into a medial (vestibular) and a lateral (cochlear) portion. The cochlear cell group is again divided into two portions by the ingrowing fibres of the corpus restiforme.

A difference is seen in the point of entrance of the nerve in the embryo from that of the adult. In the former the point of entrance is actually below the margin of the recessus lateralis, while in the latter it is continuous with it (see Fig. 3). This is due very likely to a more ventral extension of the recessus around the corpus restiforme so that the space comes in actual contact with the nerve as the structures of the region undergo more extensive development.

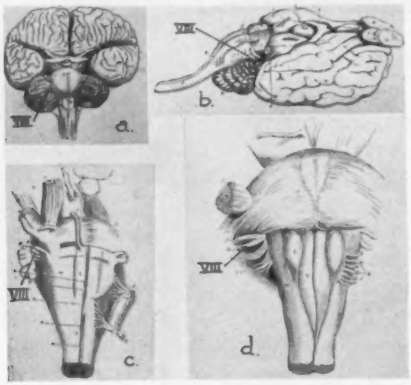

Fig. 2. The nervus acusticus and its relationships in the cerebellopontile angle in various vertebrates. (a) porpoise, (b) sheep, (c) horse, (d) elephant. Copied from Solly.

German investigators strongly favor Connheim's "cell rest" theory as the starting point of tumors, particularly those arising in the nervous system. This active cellular proliferation of the ependymal cells of the lateral recess has been pointed out as a possible source of cell displacements leading to malformations and neoformations. Schaper and Cohen[18], in a study of the developing nervous system in typical vertebrates and man, describe a zone of active mitoses in this region and suggest its connection under certain circumstances with reparative and neoplastic activity. Orzechowski[19] believed this region to be the point of origin of atypical cyst and tumor formation, even of the now recognized nerve sheath tumors, the acoustic neurinomas. Marburg[20], narrowing the region of tumor production down to the "area parolivaris lateralis," considered that gliomas

could arise from certain undeveloped neurocytes found therein. This theory is utilized by Bielschowsky[21] in explaining the origin of the largest nodule in his case of multiple ganglioglomas (gangliogliomas (ganglioglio-neuromas). While the question of the origin of purely nervous tissue tumors has evidently been overemphasized, the large variety of new growths arising from the tissues of the recess makes it tempting to accept the notion of maldevelopment as a basis for their genesis.

Development of the Os Temporale (Pars Petrosa): The pars petrosa of the temporal bone develops in the chordal or axial portion of the skull. The mesenchyme surrounding the ganglion acustico-facialis becomes condensed about the fourth week of embryonic life

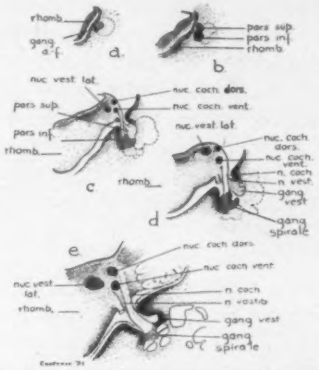

Fig. 3. Development of the ganglia and fibres of the nervous acusticus diagrammatically represented. Only the root of the n. facialis is shown. (romb.) rhombencephalon, (gang. a-f.) ganglion acustico-facialis, (pars sup. and inf.) pars superior and inferior of the ganglion acustico-facialis, (nuc. coch. dors. and vent.) nucleus cochlearis dorsalis and ventralis, (nuc. vest. lat.) nucleus vestibularis lateralis, (n. coch.) nervus cochlearis, (n. vest.) nervus vestibularis, (gang. vest.) ganglion vestibularis, (gang. spirale) ganglion spirale (cochlearis).

and during the third month is converted into cartilage (cartilaginous ear-capsules). The ear capsules (petromastoid) become ossified from four centers during the fifth and sixth months; one appears in the promontory of the medial walls of the middle ear, one roofs in the tympanic cavity and antrum, a third appears near the posterior semi-circular canal and the fourth arises near the eminentia arcuata. It is the last which forms the bony walls enclosing the acoustic ganglia and the first portion of the nerve. While these various divisions are usually coalesced after the sixth month of uterine life, the lines of junction are very vascular and in some instances may even be separable during infancy. It is possible, as Macewen[22] has suggested, that inflammation may extend through these lines of cleavage

to gain entrance into the meningeal spaces from the middle or internal ear.

A developmental process which is still in progress during infancy and childhood is the gradual obliteration of the fossa subarcuata. During this period, an opening, situated above and behind the porus acusticus internus, represents a depression or even a canal which extends into the pars petrosa under the superior semicircular canal. Occasionally, it may even approach the mastoid. A vein surrounded by a fold of pia mater sometimes extends into it. Purulent exudate has been traced from the mastoid to the cranial cavity through this canal.

If there are any peculiarities in the development of the dura mater covering the posterior surface of the petrous bone, they have not been described. Aoyagi and Kyuno[23], in their study of the arachnoidal "cell rests" in the dura, described some of them as occurring in the region of the porus acusticus internus and the fossa subarcuata. It is possible that these were embedded during the early development of the dura and arachnoid, serving in later life as a point of departure for the growth of meningiomas of the petrous ridge (Cushing[24]).

ANATOMIC ASPECTS.

The nervus acusticus is, with the exception of the first, the shortest of the cranial nerves, and probably shares honors only with the second in importance in the field of clinical medicine. Lesions of the eighth nerve cause their victims to early seek aid of the otologist and a differential diagnosis must be made between conditions involving the nerve and its end organs. The situation and course of the nerve explain the rather characteristic symptoms produced.

The eighth nerve is small, grayish in color and softer in consistency than most of the other cranial nerves*. According to Henschen[12]

Nervus acusticus	n. vestibularis	pars superior	r. ampul. sup. r. ampul. ext. r. recess. utric.
		pars inferior	r. sacc. r. ampul. post.
	n. cochlearis	ramuli spirali	

This distribution would indicate a complete functional independence of the fibres of the n. vestibularis and n. cochlearis, a feature which is not recognized in other classifications.

it is 17-19 m.m. long in the adult male, 16-17 m.m. in the adult female, and but slightly shorter in infancy. This is in accord with

*There has been some difference of opinion as to the proper grouping of the fibres constituting the two anatomic portions of the eighth nerve. The classification originally proposed by Cannieu has met with more favor than others suggested and, from an embryologic standpoint, seems to fit the situation most exactly, as the studies of Streeter[17] have shown. This classification is added herewith.

my observations. It has a greater diameter than the nervus facialis which it accompanies. It extends from the cochlear and vestibular ganglia to the recessus lateralis. It is divided into two parts: *1.* the preganglionic and *2.* postganglionic portions. The last mentioned portion is the longer of the two and in turn may be subdivided into three parts: *a.* a short intracanalicular portion lying within the porus acusticus internus, *b.* a longer free portion situated within the subarachnoid space (cisterna lateralis), and *c.* a short intrapontine portion which terminates in the vestibular and cochlear ganglia. Within the internal auditory canal, the n. facialis lies dorsal to it, but within the subarachnoid space crosses below to terminate caudal to it in the posterior pons.

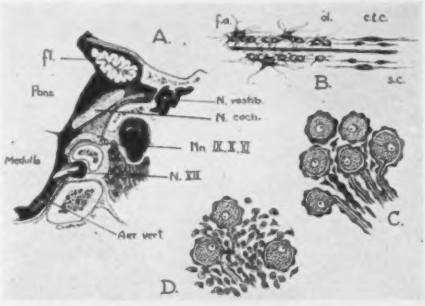

Fig. 4. Diagrammatic drawing of the histologic aspects of the nervus acusticus and its ganglia. (A) nn. vestibularis and cochlearis entering the porus acusticus internus. The lighter glial portion of these nerves makes a dome-shaped junction with the peripheral nonglial portion. (fl) flocculus of the cerebellum. (art. vert.) arteria vertebralis. (B) Cellular elements in the glial and nonglial portion of the nerve. (f.a.) fibrous astrocyte, (ol.) oligodendroglia, (c.t.c.) connective tissue cell, (s.c.) sheath cell (of Schwann). (C) Showing well ordered arrangement of the sheath cells in the ganglion spirale. (D) Confused arrangement of sheath cells in the ganglion vestibularis.

The eighth nerve, as indicated by the frozen sections of Macewen[25] (see Figs. 5 and 6) and examination of the brain stem *in situ*, takes a medialward and slightly downward and backward course. In infancy it lies almost horizontal within the skull, its medial extremity tending to assume a lower position as life advances. There is usually a slight flexure at the point of entrance to the porus acusticus internus, particularly when the bony canal takes a somewhat posterior direction from its medial to lateral limits. At the point of entrance into the brain stem it winds around the restiforme body much as the tractus opticus does about the crus cerebri (see Fig. 7). Its anterior (cephalic) fibres are those of the vestibular portion of the

nerve and enter the brain stem, not at the medullopontile junction, but at the lateral aspect of the posterior portion of the pons itself. At this point the corpus restiforme is bending sharply dorsally and caudally into the cerebellum. The more caudal cochlear fibres are divided into two portions. The anterior fibres of this group enter the nucleus cochlearis ventralis while the caudal group enter the nucleus cochlearis dorsalis.

At the point of entrance to the pons the nervus acusticus lies in close proximity to the nervus facialis which grooves it. Between the two is usually found the nervus intermedius (see Fig. 8). There is little variation in the relationship of these two nerves in the human, but often considerable in the various vertebrates. In the sheep, for

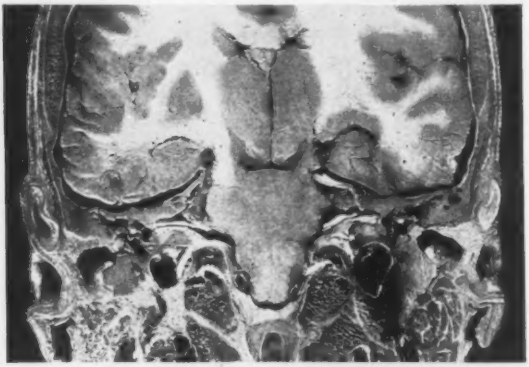

Fig. 5. Portion of a coronal section of the head, showing nervi facialis et acusticus entering the porus acusticus internus. The relationship of these nerves to the structures forming the cerebellopontile angle is also indicated. (From Macewen, "Atlas of Head Sections." James Maclehose & Sons, Glasgow, Plate 16.)

example, there is some distance between the root of the eighth and the more caudally emerging root of the seventh nerve. The seventh root leaves the pons slightly medial and caudal to that of the eighth.

In the subarachnoid space the seventh nerve lies anterior to the eighth and crosses it from below upward in this position. Arising medially and slightly caudally to the roots of the nervus acusticus, it passes anterior to it and enters the internal auditory canal in a dorsal position. In addition to this crossing of the two nerves, there is also a crossing of the fibres of the vestibular and the cochlear portion of the n. acusticus. This is due to two factors. The receptor ganglia of the pons for the vestibular portion lie more medially and cephalically than those of the auditory position. Tracing the fibres peri-

pherally, we find that those of auditory function have their origin in the cochlear situated in a plane anterior to the vestibule. This fibre crossing is not angular but occurs in a spiral fashion which may at times be made out by careful separation of the individual fibres *in situ*. The auditory fibres pass from behind forward and above those of vestibular function. The spiral twist is therefore counterclockwise on the right and clockwise on the left. As stated by Skinner[26], this crossing is usually not apparent in sections of the human nerve, although it can be distinguished in sections of this nerve in the rabbit. It is also clearly shown in the illustrations of His[15], showing the relationship of the eighth nerve in the embryo. It is likely that it is this partial spiral of the constituent fibres as well as the comparative softness of its texture that renders the nerve more suscep-

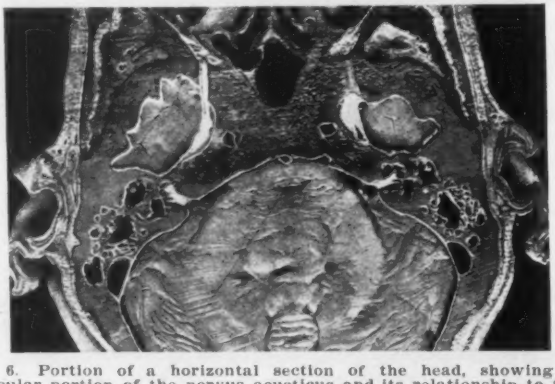

Fig. 6. Portion of a horizontal section of the head, showing intracanalicular portion of the nervus acusticus and its relationship to the end organs. (From Macewen, "Atlas of Head Sections," James Maclehose & Sons, Glasgow, Plate 40.)

tible to trauma as well as to distortion effects of "angle" tumors than the accompanying nervus facialis. Finally, it is to be remembered that there is also some twisting of the individual fibres of the cochlear portion of the nerve, so that it is most likely to suffer when pressure is put upon it by enlarging tumors.*

Blood Supply and Drainage: The arteria auditiva interna is a long slender vessel lying in the subarachnoid space and is difficult to trace

*The relationship of the individual fibres of the cochlear nerve has been considered to be of importance by Randall (The Localization of Fibres in the Acoustic Nerve, Trans. Amer. Otol. Soc., 18:292, 1928) in the explanation of symptoms produced by pressure on the nerve. The fibres from the lower turn of the cochlea surround in a spiral fashion those of the upper turns and, consequent to their peripheral situation, are the first to feel the effects of pressure by a growing tumor. According to this conception, nerve deafness for the higher tones is to be expected as an early symptom of angle tumors.

with the brain stem *in situ*. Arising from the a. basilaris it courses across the basilar surface of the pons to the region overlying the recessus lateralis where it comes in contact with the nervus acusticus. It accompanies this nerve through the porus acusticus internus and divides at the external end of the canal into its two terminal branches, the cochlear and vestibular. These in turn divide into small radicles which form the capillary network of the end organs.

The essential features to be remembered are: *1*. that the artery is a very small one, and *2*. that it lies unsupported save for its attachment to the acoustic nerve itself. It may thus be easily torn in deep exploration of the angle if the brain stem is put on too much of a stretch. It probably aids in forming the arterial supply of acoustic neuromas, its radicles being easily seen in their capsule in the fresh state.

The venous drainage consists in the formation of small veins from tributaries draining the semicircular canals and cochlea, joining at the base of the modiolus, the vena auditiva interna which terminate in the posterior part of the superior petrosal or the lateral sinuses. In some instances thrombosis of these larger channels is secondary to thrombosis of these internal auditory veins. They probably have no other clinical significance.

HISTOLOGIC ASPECTS.

As originally noted by Henschen[12] and reaffirmed by Skinner[26], the VIII nerve along with most of the other cranial nerves is divided into a glial and nonglial portion. The embryonic significance of this phenomenon has already been referred to in previous paragraphs. In the human the juncture of these two portions usually occurs as a dome-shaped evagination of the glial into the nonglial segment. In some of the lower animals this line of junction is observed to be almost a straight line.

The glial portion of the nervus acusticus usually extends to the plane of the porus acusticus internus and is more extensive in the vestibular than in the auditory portion (see Fig. 4). As suggested by Skinner, the corresponding elements in the two portions of the nerve may be tabulated as follows:

Structure	Nonglial portion	Glial portion
Perineurium	Connective tissue	Extension of pia mater
Epineurium and endoneurium	Connective tissue	Neuroglia (astrocytes of fibrous type)
Sheath cells	Cells of Schwann	Oligodendroglia

The nature of the cells in the glial portion of the nerve may be demonstrated by the use of the gold sublimate method (astrocytes) and Penfield's combined method (oligodendroglia) (see Fig. 4B). Microglia have not been observed (Skinner[26]). It is the presence of these nervous elements instead of the connective tissue of the peripheral nerves which accounts for the brainlike consistency of the nervus acusticus which usually tears in this situation when the brain is removed from the cranium.

From a histologic standpoint, it may be said that it is the peripheral nonglial portion which is concerned with neoplasms of the nerve while the glial portion is more likely the part most affected by injury and toxic or infectious agents.

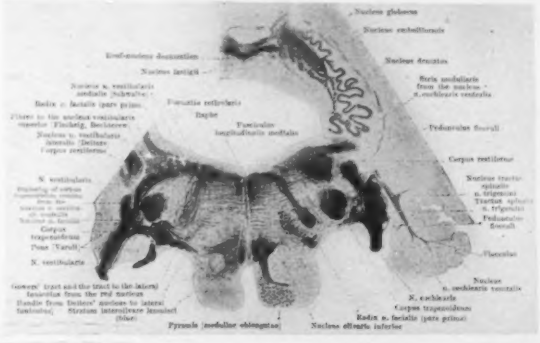

Fig. 7. Cross-section of the brain stem at juncture of the pons and medulla. The section is made slightly on the oblique so as to show the entrance of the n. cochlearis on the right and the nn. vestibularis and facialis on the left. The cisterna cerebellopontilis lies between the eighth nerve roots and the olive. (From Spalteholz, "Hand Atlas of Human Anatomy," J. B. Lippincott Co., Philadelphia, Vol. iii, p. 682, Fig. 791).

THE CEREBELLOPONTILE ANGLE.

So many of the pathologic lesions which grossly affect the nervus acusticus are located in the region adjacent to it, known as the cerebellopontile angle, that it is well to briefly consider its anatomic relationships. The history of the term has been reviewed by Cushing[27] and only mention of the various appellations given this region need be made. The following terms have been used, *recessus acusticocerebellaris* (Hartman, 1902), *cerebellopontile angle* (Henneberg and Koch, 1902), *angulus pontis* and *receptaculum pedunculorum* (Ziehen, 1903), and *acoustic region* (Ziehen, 1905). Fraenkel and Hunt[28] designated the situation as the pontomedullocerebellar space. The French have clung to the term of *tumeurs cérébello-protubérenticlles*

for new growths occupying the region. The fact remains, as Cushing has so aptly stated, that "by the time a tumor is present the so-called angle or corner has disappeared and its confines distorted beyond recognition." Because of this very temporary "angle" in case of new growths, Dandy[20] prefers to describe them as cerebellopontile tumors.

The only excuse for this tardy effort to follow in Henschen's footsteps in describing the relationships of the region is to lay some sort of a foundation for the following contributions of the series. In anticipation, it may be stated that the sequence by which the various structures are involved by new growths depends upon the tissues from which they arise and what may be a "cerebellopontile angle

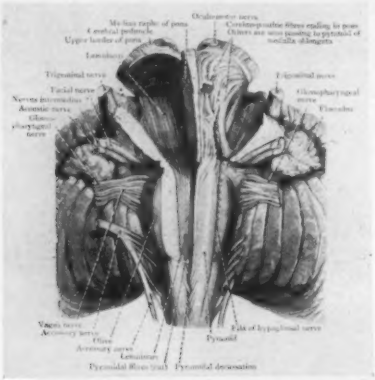

Fig. 8. The relationships of the nn. acusticus et facialis in the cerebellopontile angle. The tuft of choroid plexus normally present is not shown. (From Cunningham, "Manual of Practical Anatomy," 7th ed. New York, Wm. Wood & Co., Vol. iii, p. 495, Fig. 199.)

tumor" in one case will prove to be a "medullocerebellar angle" or a "pontomedullocerebellar angle tumor" in another. Strictly speaking, we are dealing with a more or less potential rather than an actual space. The pons and medulla fit snugly into a gently sloping depression in the basilar portion of the occipital bone. At times, when prominent, the flocculus fits into a small depression in the posterior surface of the petrous bone just caudal to the porus acusticus internus. The space lying between the brain stem, cerebellum and the dura covering the posterior surface of the petrous bone consists in *a*. a groove between the pons and cerebellum, *b*. a groove between the medulla and the cerebellar tonsil and *c*. an irregularly cuboidal space between the two. This space is bounded medially by the olive,

rostrally by the caudal border of the pons, and caudally by the cerebellar tonsil. Its floor, *in situ*, is formed by the arachnoid, crossed by the anterior inferior cerebellar artery. Its roof is formed by the white substance of the cerebellum. Its lateral wall is formed by the cortex of the tonsil and flocculus, and is covered by the fibres of the nn. vagus and glossopharyngeus. In the outer rostral angle of this roughly cuboidal space are the roots of the nervi facialis and acusticus*. The arachnoidal floor is prolonged laterally into a cone surrounding the roots of these nerves and enters the internal auditory canal (see Fig. 8). This is shown by Locke and Naffziger[30] to be a small bulbous expansion of the arachnoid around the seventh and eighth nerves. By the introduction of dyes into the arachnoidal cone about the nervus acusticus, Eagleton[31] found that the coloring material tended to go *1*. into the middle fossa of the skull with the nervus oculomotorus, *2*. about the "superior collar of the cerebellum" and *3*. along the surface of the medulla and cord†. No details are added as to the size and shape of the local arachnoidal sac itself. His observations are in accord with those of others.

Thus instead of an "angle" we are dealing with an irregular cisternal space, appearing not unlike a miniature Bedouin's tent, with the facial and acoustic nerves serving as a curving central support. The "angle" cysts following infectious processus of the middle ear and mastoid, with resultant adhesions, consist largely in the distention of this space. As Cushing has already mentioned, the trapping of fluid in this space is also accountable for the superimposed "cyst" covering cerebellopontile tumors, encountered in the usual surgical approach. He accounts for this occurrence by obstruction of the outflow of the fluid which is secreted by the tuft of choroid plexus

*This space is evidently the fossette latérale du bulbe of the French anatomists.

The method that I have employed to best advantage in demonstrating the ramifications of the subarachnoid space of this region is the injection of the space in situ with melted paraffin. In a case being autopsied the head is opened in the usual manner and the cerebrum removed by high section of the cerebral peduncles. This leaves the structures of the posterior fossa with their normal relationships undisturbed. The dura is cut along the petrous ridge until the nervus acusticus and its environs are exposed. The arachnoid is identified by gentle traction on the brain stem and the subarachnoid space carefully injected with melted paraffin with the aid of a glass syringe and needle. The paraffin is allowed to harden, following which the brain stem and cerebellum are removed intact. The finer details of the space are well shown if the injection has been successful. This is impossible if the brain has been removed before injection is attempted for the perineural sheaths have been torn away and the arachnoid itself is also often perforated. Coloring matter may be added to the paraffin if desired.

†In describing the arrangement of the local arachnoid, Boss (Topographie der Arachnoidalzisternen. Zentralbl. f. Chirur., 53:542, 1926) refers to the cisterna magna or cerebellomedullaris, the cisterna pontis medei (about the basilar artery) and the cisterna pontis lateralis. It is this last small cistern that we are concerned with in this connection, lying as it does in contact with the petrous bone and communicating with the auditory canal. It is the space involved in Bárány's syndrome (hydrops of the cisterna pontis lateralis).

which extends into the angle. The release and accumulation of this fluid has been suggested as the cause of fluctuation of symptoms which occur in tumors of the region.

SUMMARY.

The nervus acusticus and its connection with the organ of hearing has been recognized for centuries. While the details of its structure vary considerably in the various vertebrata, its general position and relationships are remarkably constant. The difference in the extent of the glial portion of its two portions is to be explained on an embryologic basis and may account for the occurrence of tumors of the eighth nerve. The peculiar structure of the nerve and the variability of related recess tissues accounts for the wide variety of neoplasms which come to occupy the cerebellopontile angle. The soft texture of its central portion and the crossing of its constituent fibres account for its frequent damage in craniocerebral injuries and susceptibility to injury and distortion by local new growths. A brief review of its essential histologic and anatomic aspects has been made. Reference is made to the region now commonly known as the cerebellopontile angle and its anatomic limits are also discussed. This contribution serves primarily as a basis for future ones dealing with pathologic and clinical considerations.

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THE NERVUS ACUSTICUS.

II. PATHOLOGIC CONDITIONS INVOLVING THE EIGHTH NERVE AND THE CEREBELLOPONTILE ANGLE.*†

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Lesions of the intracranial portion of the nervus acusticus are now recognized to be fairly common. Delayed or missed diagnoses in such cases can usually be attributed to one factor, failure to consider all the possibilities. It is the purpose of this, the second contribution in the series, to recall and briefly describe typical lesions occurring in the cerebellopontile angle which involve the VIII nerve either primarily or secondarily. Only by such an acquaintance with pathologic changes giving rise to auditory symptoms can the clinician hope to make a diagnosis early enough to give the patient any sort of chance for recovery. This contribution is limited to a consideration of the gross pathology; the histologic detail of gross lesions or of purely microscopic lesions will be considered in a succeeding paper.

TRAUMATIC CONDITIONS.

Fractures of the base of the skull are fairly common accompaniments of severe injuries to the head and the petrous portion of the temporal bone is often involved. Following injury, bleeding from the ear or a subcutaneous extravasation of blood in the mastoid region (Battle's sign) are considered to be pathognomonic of this condition. Dr. John Schaefer[1], of the Los Angeles coroner's service, informs us that fracture lines involving the petrous portion follow no characteristic course but run in every possible direction. Their distribution is probably dependent on the point of application of force producing the fracture. They occasionally pass through the porus acusticus internus. Because of its irregular shape and hard texture, there are probably no points of special weakness in the petrous bone which influence the line of fracture. Such fractures may give rise to auditory symptoms through damage to the middle ear, the end organs, the nerve or its central ganglia. It has been commonly supposed that such symptoms are due to injury to the middle or internal

*From the Department of Pathology, Los Angeles County General Hospital.
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ear but complete laceration of the VIII nerve is not uncommonly seen in cases coming to autopsy. One may reasonably assume that patients who survive a basal skull fracture may have some degree of pathologic changes in the VIII nerve. Because of its soft consistency, its flexure at the porus internus and its action in suspending the pons in the posterior fossa, it is especially liable to stretching and bruising, with resulting impairment of its function. Such injuries are probably the cause of the clinical symptoms of the vestibular apparatus as reported by Linthicum and Rand[2], who found typical cerebellopontile responses to caloric and rotatory stimulation. Furthermore, in studies on the histologic changes in the brain following head injury conducted in our laboratory, small petechial hemorrhages have been observed in the central nuclei of the acoustic and vestibular nerves. On studying the nerve cells comprising these ganglia under higher magnifications, vacuoles have been found in their nuclei and cytoplasm. It is possible that these vacuoles indicate damage to the ganglia along with the rest of the brain (generalized edema), and that more or less permanent injury may result from these changes.

The dura is so firmly attached to the petrous bone that local extradural hemorrhage is unheard of. Hemorrhage following cranial injuries is usually subarachnoid as far as the cerebellopontile angle is concerned. In cases surviving injury, it is unlikely that an accumulation of blood in the angle or its subsequent organization has very much to do with the production of auditory symptoms. At times a hemorrhage into the pons will rupture into the angle with the formation of a clot in this situation. Could a patient survive such a lesion, typical cerebellopontile angle findings would probably be elicited.

VASCULAR CONDITIONS.

In addition to hemorrhage produced by trauma, other vascular lesions are to be thought of. Meniére's disease may be dismissed as a lesion of the end organ rather than the nerve itself, although this condition has occasionally been diagnosed when in fact a tumor has been present. Thrombosis of the local vessels with resulting softening of the lateral margin of the medulla (inferior olive) and adjacent cerebellum is not accompanied by eighth nerve symptoms, unless the vertigo is due to disturbance of the terminal nuclei.

One vascular lesion should be especially considered in this connection, not only because of the auditory symptoms which it may produce, but because of the possibility of confusion which might occur

with other lesions of the region. An *aneurysm* arising from the upper portion of the vertebral artery tends to form a bed for itself in the adjacent structures by dislocation and compression, much as does a typical angle neoplasm. These aneurysms arise from either the vertebral artery itself or from its juncture with the posterior inferior cerebellar artery. Gowers[3] lists aneurysms in this situation as being seventh in order of frequency of the various intracranial arteries. Cases of this type have been described by Schultze[4] and Dandy[5], the latter finding one in the course of a suboccipital exploration.

Through the courtesy of Dr. V. L. Andrews, pathologist to the Hollywood Hospital, we have had the opportunity to study a specimen of this lesion. A summary of the history and findings in this case follows.

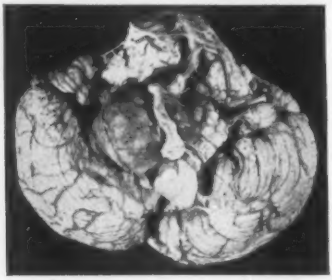

Fig. 1. Case 1. Aneurysm of the right vertebral artery compressing the structures of the cerebellopontile angle. Case of Dr. V. L. Andrews.

Case 1. Aneurysm of Intracranial Portion of Right Vertebral Artery Involving Cerebellopontine Angle Region.

History: The patient, a 45-year-old Caucasian toolmaker, had for many years complained of headaches and occasional attacks of rheumatism. Four years ago he had had a right mastoidectomy because of "ear trouble" but since has continued to complain of aching and impaired hearing in the right ear. More recently visual disturbances, unimproved by repeated fittings with glasses, and changes in temperament had made their appearance. He complained of spells of vomiting, dizziness, nervousness and malaise at the time he was first examined by his physician, Dr. Glenn English, a month before admittance to the hospital. Shortly before admittance there were times when he was unable to swallow. Early in the morning, April 11, 1931, he had complained of unusually severe headache and had

rather suddenly become semicomatose and was admitted to the Hollywood Hospital the same day.

Examination: When first seen a month previously, the only positive findings recorded were a blood pressure of 214/136, a systolic murmur at the apex and albumin and casts in the urine. He had improved on a light diet and bed rest until the night before admittance, when he again had a severe headache. In the hospital he was found to be dyspneic and cyanotic, the pupils were pinpoint, the superficial and deep reflexes were abolished throughout and he was unable to swallow. He died at 6:20 p. m., the day of admittance.

Necropsy Findings: Arising at the juncture of the right vertebral and posterior inferior cerebellar artery was a nodular appearing aneurysmal sac measuring 3.5 x 3 x 2.2 c.m. in its greatest cross-sectional diameters (see Fig. 1). It had formed a bed for itself by compressing the adjacent medulla, pons and cerebellum, giving rise to the typical distortion effects of angle tumors. The medial aspect of the sac was crossed and grooved by the left vertebral artery.

Comment: This case presents the most important gross vascular lesion of the cerebellopontile angle which results in auditory symptoms. Evidently the mastoidectomy had been done on the basis of the developing auditory symptoms. Too much emphasis cannot be laid on the importance of complete examination in cases with deafness as a presenting symptom. It would have been of interest to know what the vestibular test would have shown.

INFECTIOUS LESIONS.

Among the processes which extend into the angle and give rise to symptoms involving the eighth nerve, perhaps none are of more concern to the otolaryngologist than those secondary to infection of the middle or internal ear. We are concerned in this connection only with those giving rise to auditory symptoms or the "angle" syndrome. Worth while mentioning in this connection are localized infectious meningitis, chronic arachnoiditis with the formation of inclusion cysts, and subacute or chronic infection involving the pons and middle cerebellar peduncle. The difficulties in diagnosis are augmented by the presence of the primary condition which so frequently have already resulted in auditory manifestations. Neurologic and vestibular examinations furnish the only clue to exact localization.

Localized Suppuration in the Cerebellopontile Angle: Generalized suppurative meningitis is characterized by an accumulation of pus in the subarachnoid space. Such collections are naturally larger in the cisternal spaces at the base of the brain. While the cerebellopontile

angles are filled with exudation on such occasions, symptoms of acoustic origin are seldom elicited owing to the stuporous condition of the patient. Of more interest to us in this connection is the occurrence of *localized* collections of pus in the angle, a localized meningitis consequent upon suppuration in the middle ear or mastoid. This condition is but a stage in the development of generalized meningitis which as a rule will eventuate in death unless treatment is instituted, and usually in spite of it. Such local suppurations occur usually in one of two forms: *1.* following osteomyelitis of the petrous tip, and *2.* local extension through the internal auditory canal into the cerebellopontile angle. The first type, associated with symptoms referrable to the fifth and sixth nerves (Gradenigo's syndrome) does not produce auditory phenomena and so will not be considered here.

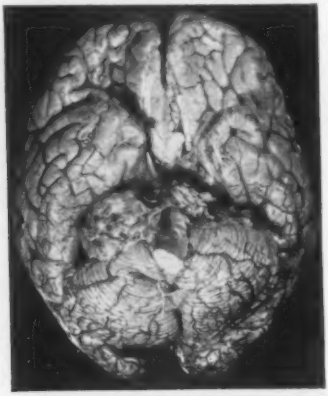

Fig. 2. Typical acoustic neurofibroma. The patient was a 49-year-old female.

In less active infections due to less virulent organisms the stages of extension are definitely prolonged. This permits reactive processes to develop so that suppuration extending into the cerebellopontile region may be localized here for a variable period. Such localized collections of pus may give rise to a more or less typical angle syndrome. While such a condition is not common, it is to be thought of when cerebellopontile angle symptoms appear in the course of chronic middle ear disease. The limitation of the process is due to developing adhesions at the periphery so that there is formed in fact a subdural abscess. According to the observations of Eagleton[9] such accumulations of infectious material are most

commonly due to necrosis of the petrous apex and provoke symptoms typical of Gradenigo's syndrome rather than those of the cerebellopontile angle. It has not been our opportunity to study such a case clinically or at necropsy.

Arachnoidal Cysts (Chronic Cisternal Arachnoiditis): A more chronic process following middle ear infection is that of a chronic arachnoiditis. In actuality the condition is not truly infectious but probably represents a toxic reaction to the adjacent local inflammatory process. The meninges become thickened, whitish and opaque. The formation of adhesions results in a trapping of the fluid in the angle, which is continually added to by the action of the tuft of choroid plexus extending out from the fourth ventricle. Rupture, with consequent refilling of such inclusions may give rise to a fluctuation of the clinical symptoms. While not necessarily confined to the angle, as Horrax[7] has suggested, the cerebellar phenomena are prominent wherever the accumulation is situated. When the angle is involved the picture is rather characteristic.

A brief resumé of a case which has been reported elsewhere in detail by Dr. I. Leon Meyers[8] is here appended.

Case 2. Arachnoidal Cyst in the Right Cerebellopontile Angle Following Acute Otitis Media and Mastoiditis.

History: L. A. C. G. H., Case 78-926. The patient, a one-year-old Mexican female, was admitted to the Los Angeles General Hospital on Oct. 14, 1929, with a fever of undetermined origin. Double myringotomy resulted in profuse purulent drainage, more abundant on the right side. Because of evident extension, a right-sided mastoidectomy was done on Nov. 17, 1929.

Examination: On neurological examination by Dr. Meyers a cervical rigidity was found. There was a tendency to deviation of the head and eyes to the left and a well marked horizontal nystagmus to the left. There was some weakness in the right arm and leg, associated with an inconstant right Babinski.

At operation, there was found an arachnoidal cyst located beneath and lateral to the right cerebellar hemisphere. It evidently represented a primary involvement of the cistern in the angle which had extended posteriorly to present itself when the region was explored. The arachnoid forming the cyst was adherent to the cerebellum and, on opening the "cyst" it was found to have no special lining aside from the local arachnoid. The cerebellum gave evidence of pressure by the cyst. The cerebellopontile angle was examined and no other pathology was demonstrated.

The child died the night following exploration. At autopsy the presence of the cyst in the angle was evidenced by a depression of the adjacent cerebellum and less so of the brain stem. The reflected arachnoid forming the wall of the cyst was well demonstrated. Pus was found in both middle ears and mastoids.

Comment: The age of the patient made adequate examination of the auditory function impossible and the presence of drainage from the ears prevented a neuro-otologic study. The typical operative and autopsy findings indicated that such symptoms must have been present. In adults, the more or less typical symptom-complex is usually presented. Pathologically the condition must be classified as an arachnoidal cyst of the cerebellopontile angle, a result of chronic arachnoiditis with adhesions.

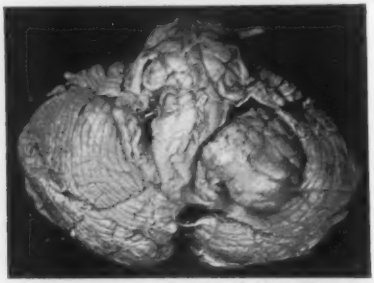

Fig. 3. Case 5. "Psammoma" arising from the tuft of choroid plexus in the cerebellopontile angle. Case of Dr. Howard Ball.

Other Inflammatory Lesions: Except for extensions of infectious processes of the meninges at the angle into the adjacent nervous tissues, direct involvement of the brain stem and cerebellar peduncles in this region is unusual. A case which deserves special mention is here appended. Its counterpart we have been unable to find in the literature. Evidently the infection had extended along the regional cranial nerves to the brain stem with resultant involvement of the regional fibre tracts. This may be another way in which infection may reach the cerebellum.

Case 3. Diffuse Inflammatory Process Involving Right Brachium Pontis and Cerebellar Hemisphere Following Chronic Otitis Media.

History: L. A. C. G. H., Case 100-036. The patient, a 32-year-old white male, was admitted to the hospital on March 17, 1930,

with the complaint of right otitis media and deafness in the right ear for 15 years. These symptoms were associated during the past month with right-sided headaches, vertigo and "paralysis" of the right side.

Examination: There was purulent drainage from the right ear, associated with total deafness in that ear. Neurologic examination by Dr. Meyers revealed an irregularity of the pupils, the right smaller than the left. There was a deviation of the eyes to the left and the head to the right. There was weakness of the right side of the face of peripheral type, and paralysis of the soft palate and partial paralysis of the muscles of the pharynx. He was dyspneic and partially aphonic. The deep reflexes were hyperactive throughout, but more so on the left side. The patient was ataxic, falling to

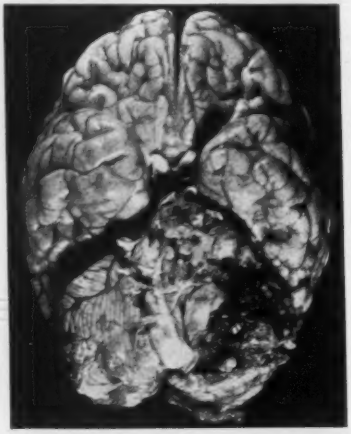

Fig. 4. Case 6. Ependymal glioma arising in the recessus lateralis and invading the cerebellopontile angle. A portion of the tumor was also found in the fourth ventricle.

the left, and past-pointed to the right. There was a well developed horizontal nystagmus on looking to the right and a feeble nystagmus on looking to the left.

Neuro-otologic examination by Dr. Isaac Jones showed all canals on the right side to be unresponsive. On the left the vertical canals were responsive, but the horizontals were atypically active, as evidenced by an *oblique* nystagmus of large amplitude.

Laboratory Findings: White blood count, 13,300; polymorphonuclears, 68 per cent; lymphocytes, 32 per cent. Negative blood Was-

sermann, positive Kahn. Lumbar puncture revealed clear fluid, with no increase in cells or globulin. Kahn and Wassermann tests were negative.

Autopsy: Examination was limited to the head. There was an obvious enlargement of the right brachium pontis and the right cerebellar tonsil was herniated into the foramen magnum. On transverse horizontal section the right brachium pontis was found to be enlarged, as well as the white substance of the right cerebellar hemisphere. The swelling of the cerebellar peduncles (inferior and middle) had resulted in local pressure on the emerging cranial nerves. On histologic examination there was found an infiltration of the

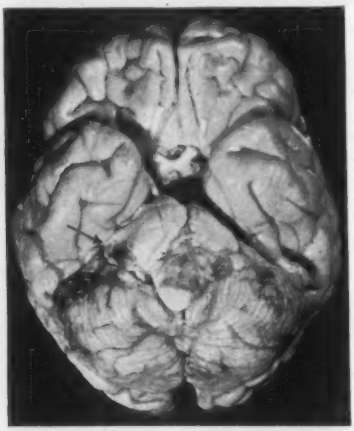

Fig. 5. Case 7. Pontile glioma, an extension of which (arrows) compressed the nn. facialis and acusticus with typical angle symptoms resulting.

peduncle and adjacent cerebellum with round cells and polynuclear cells. The involved tissue was edematous.

Comment: This unusual case apparently represented an early stage of an infectious process involving the right brachium pontis and cerebellar hemisphere. It is possible that the condition might have progressed to suppuration with the formation of an abscess had the patient survived. The cranial nerve symptoms were evidently due to pressure on their emerging roots by the swollen cerebellar peduncles on the right side.

The involvement of the cerebellopontile angle, with resultant eighth nerve symptoms by infectious processes, may be summarized as follows. In acute inflammations (suppurative meningitis), accumula-

tions of pus in the angle are but a part of a generalized process and without clinical or pathologic significance. In subacute or chronic processes with less virulent organisms, a localized meningitis with angle symptoms may occasionally occur. As a purely reactive process incident to the toxins of middle ear infection, arachnoidal adhesions may develop with loculation of fluid (arachnoidal cyst). Typical cerebellopontile angle symptoms occur with such a cyst. A case of hitherto undescribed inflammatory involvement of the middle and inferior cerebellar peduncles with angle symptoms is recorded.

Certain chronic infections, notably syphilis, attack the eighth nerve but the pathology of such conditions is of microscopic proportions and will be dealt with later. Infectious granulomata will also be discussed under the section on tumors.

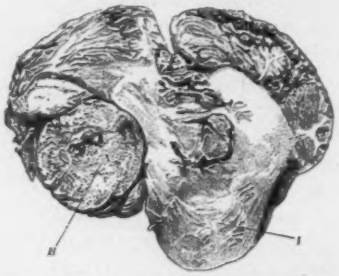

Fig. 6. Bramwell's case of metastatic tumor of the cerebellopontile angle. The distortion of the adjacent brain stem and cerebellum is characteristic. (H) tumor, (I) pons.

NEW GROWTHS OCCUPYING THE CEREBELLOPONTILE ANGLE.

There are many different types of tumors which may be found in an enlarged and distorted cerebellopontile angle.* They may arise from practically every regional tissue or structure. The classification of Henschen[9] appears to be complete in this respect.

*In this consideration of tumors of the cerebellopontile angle, we have limited ourselves to the discussion of the more common varieties or to the presentation of cases of sufficient interest to warrant their appearance in the literature. Needless to say, reports of rare and unusual tumors may be found, particularly those which are secondary. In this group may be included those arising in the region of the sella, from the accessory nasal sinuses or from the base of the skull. Bailey described a case of craniopharyngeal pouch cyst which extended into the cerebellopontile angle, unaccompanied, however, by suggestive auditory symptoms (Arch. Neurol. and Psychiat., 11:142-144, Feb., 1924). Roussy and Bazgan reported the case of a tumor, supposedly of choroidal origin but not resembling it histologically, which apparently arose from the hypophysis and extended posteriorly into the angle (Rev. neurol., 2:122-132, July, 1927). It is possible that chordomas arising from the basisphenoid and the rare osteogenic sarcomas of the base of the skull might easily provoke auditory phenomena by involvement of the eighth nerve.

A. Primary Tumors: 1. Tumors arising from the adjacent nerves, particularly the acoustic—neurinomas. 2. Tumors arising from the dura mater—meningiomas. 3. Tumors arising from the soft membranes (pia-arachnoid)—cholesteatoma. 4. The adjacent brain structures, especially the flocculus of the cerebellum, the lateral recess and the *choroid plexus*. Papillomas of choroid plexus and gliomas of the pons. 5. Tumors arising from the petrous portion of the temporal bone (including the bone itself and labyrinth).

B. Secondary and Metastatic Tumors.

To this classification of Henschen might be added the following for completeness:

C. Cysts of the Cerebellopontile Angle.

D. Infectious Granulomas: 1. Tuberculomas. 2. Gummas.

Acoustic Neurinomas: Tumors of the nervus acusticus unquestionably outnumber all others in frequency. For some reason the sheath of the eighth nerve has a propensity peculiar among the cranial nerves to develop new growths. They have appeared under various designations, such as neurofibromas, neurinomas, schwannomas, fibroblastomas and peripheral gliomas. As has been already shown by Henschen and others, their point of origin seems to be in the peripheral portion of the vestibular division. The evidence leading to such a conclusion has been reviewed in the first paper of the series[10]. That other portions of the nerve may be the seat of origin is suggested by the observation of Morelle[11].

Tumors of this type are slow in growth, and because of their point of origin may enlarge the porus acusticus internus and erode the petrous ridge with characteristic Roentgenographic manifestations. They are covered with a smooth fibrous capsule, with small vessels coursing over its surface. A typical specimen is shown in Fig. 2. The color of the tumor is reddish, grayish or yellowish, due to the appearance of the normal or degenerating tumor tissue, seen through the thin capsule. The more yellow the tissue, the softer the contents, indicating regressive changes in the tumor tissue. Some are firm and fibrous, while others are soft and friable. The irregular growth and the attempt to adapt itself to the adjacent structures gives rise to a nodular and irregular surface. The growth is located in the subarachnoid space, so that at its margins the tent-like reflexions of this membrane contain accumulations of fluid such as has been described by Cushing[12]. From the posterior operative approach, this cyst-like fluid accumulation overlies the tumor and hides it from view.

The tumor varies greatly in size. Small nubbins, 1 to 2 c.m. in diameter, are occasionally encountered at necropsy in an otherwise

negative intracranial space. After wide decompressive operations have been performed the tumor may assume unusual proportions. The question of distortion of adjacent structures with production of symptoms by the enlarging tumor will be made the subject of a later contribution.

Of the various types of tumors arising in the cerebellopontile angle, the acoustic neurofibromas outnumber all others combined. According to Tooth[13] they compose four-fifths of all extracerebellar tumors (24 out of 30). These 24 cases were found in a total verified list of 258 intracranial new growths. Cushing[12], in a series of 468 verified cases of intracranial tumor, found 42 cerebellopontile

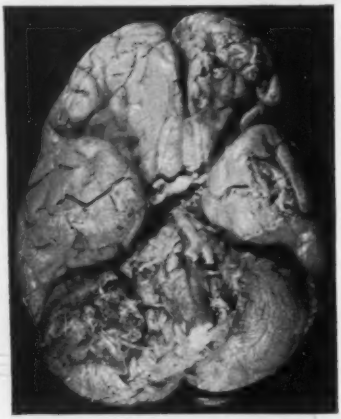

Fig. 7. Case 8. Large "epithelial" tumor in the right cerebellopontile angle. The alveolar arrangement of the columnar epithelium suggests the possibility of its origin in the structures of the internal ear.

angle growths, 30 of which proved to be acoustic neurofibromas*. Henschen[9] described 28 cases of "angle" and basal tumors, 13 of which were acoustic in origin. In this hospital, in a series of 6,000 consecutive autopsies, 100 cases of intracranial tumor were found (1.6 per cent). Of this number, five (5 per cent) were located in the cerebellopontile angle. Three of these were neurofibromas, one a meningioma and one a tuberculoma. This proportion of angle tumors to all intracranial tumors (5 per cent) probably does not represent their real frequency for a much larger proportion of angle

*Cushing's latest figures show that, of a series of 2,023 verified intracranial tumors, 176 proved to be acoustic neurinomas (8.7 per cent) ("Intracranial Tumours," Springfield and Baltimore, Charles C. Thomas, p. 8, 1932).

tumor cases temporarily recover and leave the hospital, which is not true, for example, of the more malignant gliomas. Considering a group of 185 verified intracranial tumors comprising the Brain Tumor Registry of the local Pathological Society, 26 cases (14 per cent) of cerebellopontile angle tumor are listed. Their histologic nature is shown in the following table.

TABLE I.

Tumors of the Cerebellopontile Angle as Listed in the Brain Tumor Registry of the Los Angeles Pathological Society.

Nature of the Tumor	*Per Cent*	*Number*
Acoustic neurofibromas	57.7	15*
Meningiomas	7.7	2**
Tuberculomas	7.7	2
Cholesteatomas	3.8	1
"Psammoma" of the choroid plexus	3.8	1
Ependymal glioma	3.8	1
Pontile glioma (neuroglioblastoma)	3.8	1
Metastatic tumors (melanosarcoma)	3.8	1
Unclassified tumor (internal ear?)	3.8	1
Aneurysm	3.8	1
Total		26

Meningiomas: Tumors of meningeal origin formerly designated as dural endotheliomas, now known as meningiomas (Cushing) or meningeal fibroblastomas (Penfield), which arise from this region present no essential difference from those in other situations. They are reddish, encapsulated and somewhat lobulated tumors located slightly more posteriorly than the usual position of an acoustic tumor. They frequently erode the petrous ridge and may be intimately attached to it. Cushing's conception of the origin of this tumor is that they arise from arachnoidal cells which are embedded in the dura mater. The irregular surface of the petrous bone perhaps favors this process. At any rate, as Aoyagi and Kyuno[14] have shown, arachnoidal cell groups are to be found in this situation. The clinical picture is that of a typical cerebellopontile angle tumor. In the two cases in our series, the tumors were too small to provoke symptoms.

Cholesteatomas: In addition to the aforementioned neoplasms, pearly tumors or cholesteatomas seem to have a predilection for the base of the brain and are occasionally found in the cerebellopontile angle. Such tumors are supposed to have their origin in epithelial rests of the arachnoid and are probably to be classified with tumors of dermoidal type. Cases of cholesteatomas in the angle are de-

*Of this number, two were small symptomless nodules lodged in the porus acusticus internus.

**Both of these meningiomas were small and symptomless, arising from the dura in the region about the auditory nerve. They would likely be classified as meningiomas of the petrous ridge.

scribed by Henschen[9], Tooth[13], Ballance[15] and Kaufman[16]. Cholesteatomas are not to be confused with cholesteatomatous masses formed in the middle ear, the result of chronic suppurative processes.

A case of an "angle" cholesteatoma is found in our local Brain Tumor Registry, having been successfully removed by Dr. George Patterson. A short resumé of the case is here appended.

Case 4. Cholesteatoma of the Left Cerebellopontile Angle.

History: L. A. C. G. H., Case 31-864. The patient, a 56-year-old housewife, was admitted to the hospital on Aug. 9, 1929, with the complaint of tinnitus and deafness in the left ear and pain in the left side of the head for five years, unsteady gait and weakness and numbness of the left side of the body for two years, difficulty in swallowing for one year and double vision for three months.

Fig. 8. Case 9. Tuberculoma of the left hemisphere of the cerebellum attached to the dura of the cerebellopontile angle. Arrows outline the limits of the tumor.

Examination: The patient was a rather obese white female of middle age, showing no evidence of marked discomfort. The margins of the optic discs were hazy on the nasal sides. There was a horizontal nystagmus on looking to the side, more marked to the right. Some internal strabismus was noted on the left side. There was a weakness of the left side of the face of peripheral type, associated with hypesthesia on this side. There was a weakness of the left palatal fold and sluggishness of the pharyngeal reflex on the left. The deep reflexes were increased on the left side; bilateral Babinski more marked on the left was also observed.

The patient was totally deaf in the left ear with loss of air, and retention of bone conduction. Weber was lateralized to the right. Neuro-otologic examination showed the vertical canals to be functionless on the left with an atypical response to stimulation of the

horizontal canal. On the right response was normal to stimulation of the horizontal canal and atypical to that of the vertical canals. Roentgenograms of the skull were negative.

Operation: On the diagnosis of a left cerebellopontile angle tumor a suboccipital exploration was attempted on Aug. 15, 1929, by Dr. Carl W. Rand, but owing to the patient's critical condition this had to be abandoned. The wound was re-explored a few weeks later by Dr. George Patterson and on opening the dura and retracting the left cerebellar hemisphere, a shining nodular tumor, having the typical mother-of-pearl appearance, was found filling the left cerebellopontile angle. The capsule was split and the larger portion of the soft shining pearl gray contents of the tumor was removed. As much of the capsule as could be resected was also removed. The total amount of tissue removed corresponded to a mass about 3 c.m. in

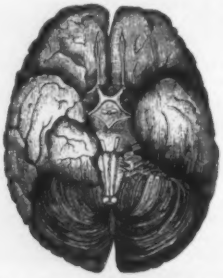

Fig. 9. Drummond's case of gumma of the cerebellopontile angle arising from the adjacent cerebellar peduncle.

diameter. Histologic examination revealed the characteristic cellular strands forming a loose reticular structure having the appearance of a honeycomb. No cholesterin crystals were observed.

Following the operation there was a complete paralysis of the left side of the face. Aside from this the postoperative recovery was uneventful. She remains well as far as can be determined.

Comment: This is a rather typical case of cerebellopontile angle cholesteatoma. It is to be regretted that more tumors in this situation are not as easily handled surgically*.

Tumors of the Glioma Group Comprising the Cerebellopontile Angle: Not infrequently new growths having their origin within the tissues of the brain stem or recessus lateralis extend into the "angle"

*Cushing describes a case in which a left cerebellopontile cholesteatoma was exposed and removed surgically. At a previous operation, an extension of this tumor into the middle fossa had been removed. (See "Intracranial Tumours," pp. 99-102.)

and give rise to typical symptoms. Among such tumors are ependymal gliomas, papillomas of the choroid plexus and gliomas of the pons.

Papillomas of the choroid plexus arise from that portion of this structure which extends from the fourth ventricle through the recessus lateralis. In the strictest sense of the term they do not belong to the glioma group. Cushing[12] described such a case as an encapsulated globular mass which had been removed *in toto* at operation. It proved to have the characteristic papillary structure on histologic examination. Another case is reported by Devic, Grandement and Puig[17]. We have not encountered such a case in our series.

A rather rare tumor originating in the tuft of the choroid plexus in the angle is described by earlier investigators as a "psammoma."† While in no way related to the papillomas of the plexus, and certainly not to be confused with any of the gliomas, we have recently had an opportunity to study such a case and for the sake of convenience a brief description of its pathology is included here.

Case 5. "Psammoma" Arising from the Choroid Plexus in the Left Cerebellopontile Angle.

The specimen in this case was brought to us by Dr. Howard Ball from the San Diego County General Hospital. It has been kept in the pathology laboratory as a museum specimen and up to the moment, no history of the case is available. The tumor, measuring 3.5 x 3 x 2.2 c.m. in greatest diameters, was a grayish, somewhat nodular encapsulated growth situated in the left cerebellopontile angle (see Fig. 3). It was but loosely attached to the structures of the region and could readily be lifted out of its bed. Its under-surface at the point of its attachment in the angle was granular in appearance and friable in structure. Histologically, it presented characteristics which were typical of meningiomas in other situations. Whorls of fusiform cells, some of which contained pinkish-purple "psammoma" bodies, were abundant.

Comment: This unusual tumor is the first of its kind to come to our attention in this situation in a series of 185 verified intracranial tumors. It was small in size and, had it been exposed at the

†This tumor is evidently very rare. We have been able to find only one reported case in which the tumor occupied the cerebellopontine angle. It was described by Sutton (The Lateral Recessus of the Fourth Ventricle; Their Relation to Certain Cysts and Tumours of the Cerebellum and to Occipital Meningocele. Brain, 9:352, 1887), small tumors being found in both cerebellopontile angles. Judging from the histological appearance in our case, these tumors have a structure practically identical with the meningiomas (dural endotheliomas). If such is the case, they must have their origin from arachnoidal cells within or closely associated with the regional tufts of the choroid plexus. It is evident that in addition to the true papillomas, one may encounter "psammomas" and cysts of this lateral extension of the choroid of the fourth ventricle.

operating table, complete removal would almost certainly have been accomplished.

Tumors arising from the *ependyma* of the recessus lateralis are not common, but a few cases have been described, as for example that of Henschen[9]. Cushing[12] also described a tumor in this situation, believed to be of ependymal origin. Barré, Alfandary and Stoltz[18] reported a case of ependymal tumor arising in the fourth ventricle and extending into both cerebellopontile angles. A personally studied case proved to be of interest, in that an ependymoblastoma arising in the recess came to occupy both the fourth ventricle and the cerebellopontile angle.

Case 6. Ependymoblastoma of the Fourth Ventricle and the Right Cerebellopontile Angle.

History: L. A. C. G. H., Case 137-750. The patient, a 20-year-old white female, was admitted to the hospital on Dec. 30, 1930, with the complaint of pain in the suboccipital region for two years. Nausea and vomiting had occurred for two months, and vertigo, ataxia, deafness and tinnitus in the left ear and blurring of vision had ensued three weeks prior to admittance.

Examination: Aside from rather severe headaches and blurring vision, the patient, a well nourished and well developed young woman, gave no evidence of acute illness. There was tenderness of the suboccipital region to pressure, especially on the left side. A slight bilateral exophthalmus, more marked on the right, was observed. An extreme degree of choked disc with recent hemorrhage was present. An atypical and inconstant nystagmus was found. Visual acuity was definitely lowered, especially on the right. Sensibility was decreased in the left face and the left corneal reflex was sluggish. Ataxia and hypotonia were present in the extremities of the left side. The deep reflexes were increased on the left along with a suggestive left Chaddock and Babinski.

Special examination of the ears revealed a loss of response to whispered voice. Air conduction was not present on the left; Weber was not clearly lateralized. A caloric test revealed a delayed and atypical response from the vertical canals to douching on the left side, while the horizontal canal was normal in reaction, as were all canals on the right.

On the presumed diagnosis of a posterior fossa tumor, probably involving the left cerebellopontile angle, a suboccipital exploration was performed on Jan. 1, 1931, by Dr. Carl W. Rand. A soft, reddish, vascular tumor tissue was found enveloping the nerves on the

left side, a small fragment of which was removed for histologic study. The patient did poorly after operation and succumbed on the third post-operative day following acute onset of bulbar palsy.

Necropsy: A tumor mass was found enveloping the posterior nerve roots on the left side and extended up through the incisura of the tentorium into the middle fossa on that side (see Fig. 4). Sections of the brain stem and cerebellum revealed the tumor to be in the fourth ventricle as well. The tumor tissue was grayish in color, marked with hemorrhagic spots. Microscopic sections demonstrated perivascular pseudorosettes characteristic of an ependymoblastoma. Mitotic figures indicated a malignant propensity.

Comment: This unusual case represents one of the pathologic possibilities as far as new growths involving the angle are concerned. Had the patient survived the operation, at which time the nature of the tumor had been discovered, it would have been of interest to note the effect of Roentgen ray therapy. Surgical removal was obviously impossible.

Tumors arising within the brain stem proper, *gliomas* of the pons and cerebellar peduncles do not always give rise to cerebellopontile angle symptoms. This occurs only when the growth is so situated as to compress the emerging roots of the nervi facialis and acusticus. Auditory symptoms are practically always produced by pressure on the roots and not by a disturbance of the internal mechanism of the pons. Buckley[19], in a series of 25 gliomas of the pons, described six in which auditory symptoms were observed. Such a tumor should be thought of when typical cerebellopontile angle symptoms of short duration occur in adolescents and young adults. The brain specimen in such a case has recently been studied in this laboratory.

Case 7. Pontile Glioma Provoking Typical Cerebellopontile Angle Symptoms.

History: L. A. C. G. H., Case 159-980. The patient, a 28-year-old female of German extraction, was admitted to the hospital on May 19, 1931, with a tentative diagnosis of syphilitic or tuberculous meningitis. She had developed double vision rather suddenly six weeks prior to admittance, followed two weeks later with right-sided headaches, tinnitus in the right ear, blurring of vision and numbness of the oral mucous membrane on the right side. On several occasions nausea followed by vomiting had occurred. The symptoms persisted and were inclined to grow progressively worse.

Examination: The patient was very obese and was found to be somewhat drowsy. The optic disc showed no evidence of pressure.

There was a spontaneous horizontal nystagmus in both directions, but more marked on looking to the right, and a rotation nystagmus on looking up or down. There was a definite weakness of peripheral type affecting the right lateral rectus muscle and the right side of the face. Pharyngeal reflexes were sluggish on the right side. The deep reflexes were increased and the superficial reflexes decreased on the left, associated with a Babinski and Chaddock on that side.

The patient was deaf to air conduction on the right side. On douching the ears, the vertical canals on the left and all canals on the right failed to respond.

On the diagnosis of a right cerebellopontile angle lesion, a suboccipital exploration was done by Dr. George Patterson. What was considered as a tumor arising from the pons was found in the right "angle." The seventh and eighth nerves were found winding about a small reddish nubbin protruding from the pons. The patient did poorly during the operation and respirations ceased shortly after the wound had been closed.

Necropsy: At autopsy a diffuse swelling was found involving the right side of the pons (see Fig. 5). A nodular protrusion was found at the point of emergence of the nervi facialis and acusticus. The sixth nerve likewise was embedded in the mass. On cross-section, it proved to be a gelatinous hemorrhagic tumor mass occupying the right side of the basilar portion of the pons and involving the left to some degree. The tumor proved to be a malignant glioma (neuroglioblastoma).

Comment: This is a typical example of a pontine tumor with a short clinical course. The vestibular findings were typical of an angle lesion which was considered before operation to be an arachnoidal cyst. An early fatality, as in this case, is the usual outcome of exploratory operations.

Other Types of Tumors: Among the various new growths found in this situation are mentioned teratomas (Kato[20]), epitheliomas (Bruns[21]) and metastatic tumors. Bramwell[22] illustrated a case of metastatic tumor of the cerebellopontile angle which resulted in typical distortion of the brain stem (see Fig. 6). A specimen came to our observation recently in which one of many metastatic nodules of melanomatous nature had developed within the cerebellopontile angle. Henschen[9] describes several cases of "sarcoma" of the cranial base which invade this region. What appears to be a similar tumor is described by DeMolé[23].

A group of tumors which has been given but little attention because of their rarity are those arising from the middle ear or petrous

bone and extending into the angle. Meningiomas of the petrous ridge have already been referred to. Other types classified as epitheliomas or sarcomas have been described (see Cushing[12], pp. 234-235). A rather unusual case which we have had the opportunity of studying merits a short report.

Case 8. Large Encapsulated Tumor of Epithelial Type in the Right Cerebellopontile Angle.

History: L. A. C. G. H., Case 70-181. The patient, a 57-year-old housewife, admitted to the hospital on July 23, 1929, had complained of tinnitus in the right ear for seven years, increasing deafness on the right, paralysis and numbness of the right side of the face for five years and weakness of the right side of the body for four years. For two years she had had recurrent attacks of vomiting and loss of equilibrium. Failing memory and double vision had been present for six months. Aside from a transient attack of hemiplegia on the right side ten years before admittance, the past history was essentially negative.

Examination: The patient was rather obese and because of impairment of memory was unable to give any clear account of the details of her illness. A marked impairment of the olfactory sense, especially on the left, was found. The pupils were equally dilated, reacting sluggishly to light. A bilateral choked disc with 4 diopters elevation was found. The visual fields were concentrically contracted, especially so on the left side. There was an internal squint on the left. A decrease in sensibility and an infranuclear type of weakness of the right side of the face was found. The uvula was deflected to the right and pharyngeal reflexes were sluggish on that side. There was marked ataxia and hypotonia of the right extremities. The deep reflexes were exaggerated throughout and a well defined Babinski was present on the left, with a suggestive extension response on the right.

The patient was totally deaf to air conduction in the right ear, and Weber's test showed a lateralization to the left. Caloric tests revealed all canals on the right and the vertical canals on the left to be functionless.

Roentgenograms of the skull were reported to be negative.

Course: On a diagnosis of right cerebellopontile angle tumor a suboccipital exploration was attempted on Aug. 8, 1929, by Dr. Carl W. Rand. Because of the patient's critical condition, this had to be abandoned before the dura could be opened. The patient had a stormy postoperative course, although her condition remained about

the same until the time of her death, when a sudden change for the worse brought about her demise on Sept. 17, 1929.

Necropsy: A large encapsulated tumor mass was found in the right cerebellopontile angle, more posterior in situation than the usual acoustic neurinoma (see Fig. 7). The growth had either arisen from or eroded the posterior portion of the petrous ridge so that bony fragments came away with the tumor. On cut section numerous small smooth-wall cystic spaces were observed, which gave the tissue a spongy appearance.

Histologically, the tissue had a uniform alveolar arrangement*. The spaces were lined with columnar epithelium whose symmetry indicated its benign nature. It evidently did not have its origin in the choroid plexus and may have arisen from the epithelial structures of the internal ear.

Comment: This rather unusual tumor had an unusually long clinical course. It could probably have been successfully treated surgically had the patient's condition permitted. Its exact nature or point of origin is still a question.

Infectious Granulomata: Certain of the chronic infectious diseases, especially tuberculosis and syphilis, may elaborate granulomatous masses within the cranial cavity. Tuberculomas actually arising within the cerebellum not infrequently compress the adjacent nerve structures and clinical symptoms of angle tumors often result. The following case will serve to illustrate this fact.

Case 9. Tuberculoma of the Left Cerebellar Hemisphere with Extension Into the Cerebellopontile Angle.

History: L. A. C. G. H., Case 274-863. A 16-year-old Mexican boy was admitted to the hospital on Aug. 15, 1927, with the complaint of nausea and vomiting, left-sided suboccipital headaches, ringing in the left ear and frequent epistaxis, all of six weeks duration. Aside from a previous history of pleurisy, his past history was essentially negative.

Examination: The patient was a rather poorly nourished male, who held his head tilted to the left side (occiput back and to the right). The pupils were unequal, the left being greater than the right. There was a horizontal nystagmus on looking to the right. A left internal squint was present. The discs were bilaterally choked but degree of elevation was not recorded. The left side of the face was less sensitive than the right. There was a paralysis of the left

*A more detailed description of the histology of this tumor together with illustrative photomicrographs will be presented by Dr. Ernest Hall in a later communication.

side of the face of infranuclear type. The auditory acuity was decreased but not lost on the left. Vestibular examination showed all canals on the left and the vertical canals on the right to be unresponsive. A minor degree of ataxia was present in both arms and the grip in the left hand was weaker than that on the right. A bilateral ankle clonus was present and all the deep reflexes were increased. Some stiffness of the neck and a bilateral Kernig suggested meningeal irritation.

Roentgenograms of the skull and chest revealed no abnormalities of any sort.

The blood Wassermann was negative and the spinal Wassermann strongly positive. The spinal fluid was clear and under normal pressure 30 lymphocytes per c.m.m. were found.

Course: The patient remained in the hospital for two weeks and left against advice. He died rather suddenly at his home and the city coroner performed an autopsy to determine the cause of death.

Necropsy: A tuberculoma the size of a small walnut was found in the left cerebellar hemisphere and was adherent to the dura mater in the angle (see Fig. 8). Extension into the cerebellopontile space had given rise to auditory symptoms along with those referable to other cranial nerves.

Comment: It is evident that the differential diagnosis of a cerebellopontile angle lesion is not always easy. While the acoustic neurinomas are the most common, a surgeon exploring this region may encounter any one of a wide variety of lesions. Another patient in this hospital was operated upon for an angle tumor and the bit of tissue removed from an almost inaccessible growth proved to be tuberculous in nature. Tuberculomas are to be thought of in young individuals when a typical angle syndrome is encountered. Such a case with a large tuberculoma actually situated in the angle was described by Auvert[24] in 1851.

Gummas arising from the cerebellar peduncle have been reported by Rosenstein[25] and Cushing[12]. A rather interesting case is illustrated by Drummond[26] (see Fig. 9). Similar clinical manifestations may occur in a diffuse gummatous involvement of the basilar meninges, as has been illustated by Oppenheim[27]. In these days with more active treatment of syphilis a gumma in this situation must be a pathologic rarity.

Parasitic Cysts: Cysticercus cysts have been found in the angle, particularly in European countries where the disease is not so uncommon. Such cysts are usually small and not likely to provoke auditory symptoms. Cushing[12] refers to three reported cases (those

of Hildebrand, Schlisinger and Wollenberg) in which such cysts were mistaken for angle tumors.

It is evident that this short review of the pathological anatomy of the cerebellopontile angle is scarcely more than a list of the lesions which may be found therein. A great variety of conditions may provoke the typical cerebellopontile angle syndrome in which auditory and vestibular manifestations are the most conspicuous. This group of lesions are nothing if not impressive from the standpoint of the otologist who deals daily with patients complaining of tinnitus, unilateral impaired hearing, vertigo and loss of equilibrium. Those presenting no external evidence of a causative factor should be studied with an intracranial lesion in mind.

Description of the histologic appearances of the various lesions mentioned and the pathologic physiology of such lesions are reserved for future communications.

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THE NERVUS ACUSTICUS.

III. THE PATHOLOGIC ANATOMY OF ITS NEOPLASMS.*

DR. LEO J. ADELSTEIN, Los Angeles.

Doubtless it has appeared to many members of the Study Club that an apparently disproportionate amount of time is being spent on the neoplastic affections of the VIIIth nerve; and the question naturally arises why this division of the subject should be so important from the standpoint of the otologist, and also why the neurosurgeon feels that a joint handling of the problem is to the mutual benefit of all concerned.

In answer to the first question, it is safe to say that tumors of the VIIIth nerve are relatively common. Horrax[1] has conservatively estimated that they comprise approximately 10 per cent of all brain tumors. In Cushing's series of verified intracranial tumors to Jan. 1, 1929[2], the tumors of the nervus acusticus comprise about 8.5 per cent of the entire group.

The second question is best answered by recognizing that the otologist is usually the first to see these cases. The patient complaining of tinnitus and deafness consults his ear specialist first, and an immediate differential diagnosis between an end organ lesion and a true nerve affection is imperative if suffering and even loss of life is to be prevented.

Lastly, when it is appreciated that of all the types of intracranial tumor, the tumors of the nervus acusticus are most amenable to surgical interference, further stimulation to earlier diagnosis and treatment is added. Of a relatively benign character, these tumors, whose main effects are due to pressure on adjacent vital structures rather than to invasion, offer an excellent prognosis both as to operative recovery and continued years of useful life.

It is our purpose in this short communication to discuss the gross pathologic anatomy of the neoplastic diseases of the VIIIth nerve and the accompanying perverted physiology induced by the presence of such lesions.

The monograph of Cushing[3] in 1917, collecting 30 personal cases, was the first American effort to emphasize the true clinical picture

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as well as the pathologic anatomy of the so-called "angle syndrome" produced by VIIIth nerve tumors. This had been preceded by Henschen's memorable work[4] in 1910, which really was the pioneer effort; he collected 136 cases and described a characteristic symptomatology that leaves little to be added even today.

That there never has been a dearth of good observers, even long before clinical medicine was within the sphere of the experimental method, is well exemplified by reading the notes of one John Abercrombie[5], who in 1834 described, without naming it as such, what certainly must have been a rather typical VIIIth nerve tumor giving rise to an angle syndrome. Allow me to quote verbatim:

"A woman was seen complaining of severe headache, constant vertigo, nausea, occasional vomiting, pain and deafness in the left ear, and the left eye somewhat affected; after several months the headache increased, with occasional paroxysms of coma, and she died at last rather suddenly, having been for a day or two affected with an extensive erysipelas of the head and face.

"The morbid appearances included a remarkable tumor under the base of the brain on the left side, resting on the petrous portion of the temporal bone. It consisted of three portions; the anterior was the size of an egg, and composed of a spongy vascular substance; the posterior portion was half the size of the former and of similar structure, but firmer; the middle portion was the size of a walnut, of a white color and nearly cartilaginous structure. The petrous portion of the bone on which the tumor rested was absorbed to the depth of half an inch."

The pathologic anatomy of intracranial tumors to be complete should include, according to Cushing[3]: *1.* The gross appearance and characteristics of the neoplasm, including the secondary alteration of the meninges and brain which it produces; *2.* the histological nature of the lesion; *3.* the degenerations which occur in the conducting pathways and their end stations.

In his dissertation in 1910 Henschen laid stress on the probable peripheral seat of origin of these acoustic tumors, due to the apparent excess of sheath cells in the vestibular portion of the ganglion, which explanation has been duly amplified by Courville in the earlier part of this symposium in his discussion of the developmental and structural aspects of the nervus acusticus[6].

Suffice it to say that, due to the peripheral origin of these tumors, stress was laid by Henschen upon what had been observed by many: namely, that the porus acusticus internus is commonly occupied by

a process of the tumor which may serve to enlarge it. The suggestion followed that this might prove of some diagnostic significance, and in 1912 he demonstrated that the dilated internal meatus might be shown by the X-ray. This, however, was not sufficiently appreciated until the advent of the Bucky diaphragm in 1921, which materially aided in revealing this now characteristic finding. It has been our good fortune to remove the pyramidal bone in a case of VIIIth nerve tumor at autopsy and X-ray it together with the attached process of tumor filling and widening the canal (see Fig. 1). This was done at the suggestion given by Cushing[3], who lamented the fact that in none of the autopsied cases in his series to 1917 had an examination of the pyramidal bone been made.

The Gross Appearance of Acoustic Tumors: These tumors are oval to round in shape and as they undergo enlargement, tend to conform to the shape of the tentorium above, the pyramidal bone later-

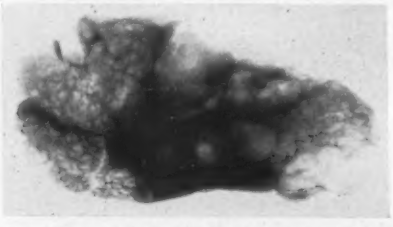


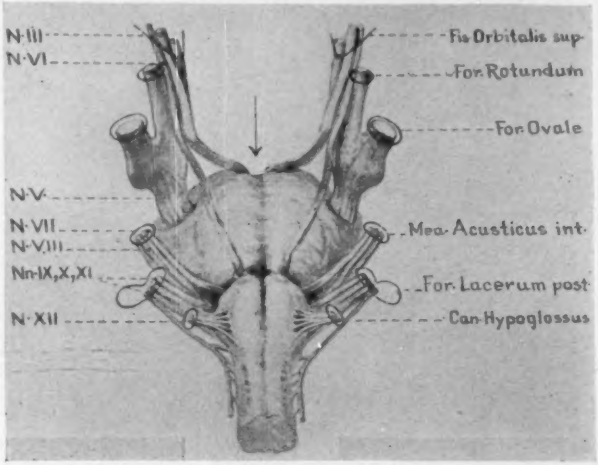
Fig. 1. Roentgenogram of pyramidal bone—actual size—showing tumor widening canal.

ally, the base of the posterior fossa below, and medially to crowd the cerebellum, brain stem and cranial nerves.

In the fresh state the specimen appears yellowish to gray. The yellower the color, which indicates extensive fatty degeneration, the easier is the tumor of removal. The more grayish, the more fibrous is the consistency of the tumor, and the more difficult is its removal. The size of these tumors vary from 2½ c.m. to 7 c.m. in length in one of the Cushing series, the average being about 3½ c.m.

Relations to the Meninges: That VIIIth Nerve tumors are not only extracerebellar and subdural, but are contained within an arachnoid capsule has never been fully appreciated. During life when the spaces are distended with fluid, this relationship is easily discernible and accounts for the cystic cap so often seen lying on the posterior aspect of the tumor and which must be opened before attacking the lesion itself.

Relation to the Cranial Nerves and Brain: The involvement of vital structures by the encroachment of a neoplasm in the small cerebellopontile angle is well known and is best illustrated by the equally well known diagrams (see Figs. 2 and 3), illustrating[5] how cranial nerves, cerebellum and brain stem share equally the effects of pressure. The cranial nerves from the fifth to twelfth are in close proximity to one another in the angle, and naturally a growth arising from the eighth nerve will in turn involve the major portion of this large group; particularly the fifth and seventh, the others to a usually lesser degree. The sixth is probably involved in vascular strangulation by lateral branches of the basilar artery[7]. The distortions of the

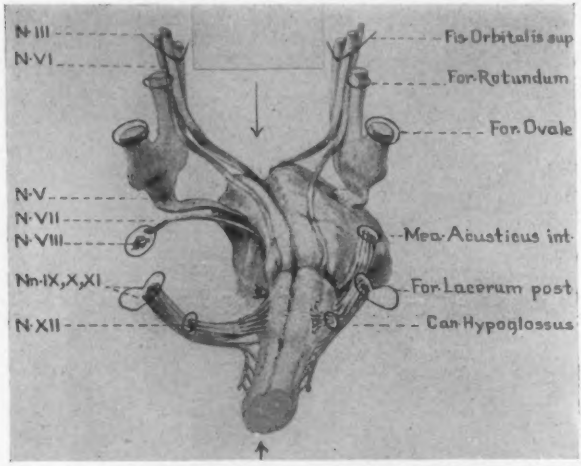


pons, medulla and cerebellum are constant and may be enormous, so that the whole bulb may be found pushed to the opposite side. The continued pressure of the tumor in turn produces a blocking of the fourth ventricle and aqueduct of Sylvius, resulting in a condition of internal hydrocephalus, with choking of the optic discs, both common findings in this type of intracranial tumor.

The histologic picture of a tumor of the VIIIth nerve has come to be recognized as a neurofibroma or, more commonly, a neurinoma (Verocay[8]). These terms have come to have a common usage and are synonymous. The nomenclature has included terms ranging from a steatoma of the older writers to a fibroblastoma of Borst. Of all the names designated, acoustic fibroneuroma or neurinoma is prefera-

ble. The histology of these lesions will be gone into more in detail in a later paper in the symposium.

Up until 1917, all tumors of the cerebellopontile angle, including cholesteatomas, papillomas, meningiomas, gliomas, as well as acoustic neurinomas, were looked upon more or less as one group, and little distinction was placed upon specific types of tumors arising in this region. Cushing, with his characteristic fortitude, declared the "time ripe for special studies of special tumors in special locations if surgical treatment was to be perfected for these difficult lesions."



Figs. 2 and 3. From Cushing's "Tumors of the Nervus Acusticus," W. B. Saunders Co., Philadelphia, showing the normal relationships of the cranial nerves contrasted with the distortion produced by a right acoustic tumor.

That this was to be a most successful point of departure in the study and treatment of specific types of intracranial tumor, is only now fully appreciated. That the surgical removal of these acoustic tumors is accomplished at the Brigham Clinic with an operative mortality of slightly over 4 per cent has fully justified all the pioneer efforts of Cushing and his school of neurosurgery, and no doubt has exceeded even the fondest expectations of the "Chief." However, what is to be stressed again is the early recognition and diagnosis of these lesions, and no one is in a better position to gain first hand information than the otologist in his daily practice, who must see that the unfortunate patient with tinnitus and deafness receives a

thorough and exhaustive examination at once, in order to rule out the possibility of a tumor of the nervus acusticus.

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THE NERVUS ACUSTICUS.

IV.—HISTOPATHOLOGY OF THE VIIIth NERVE.*†

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The pathology of the VIIIth Nerve is highly specialized and for the most part beyond the scope of the general pathologist. Circumstances not altogether under the control of the author have caused him to undertake such a study. Materials in the way of microscopic sections illustrative of the histology of many of the gross lesions presented in the preceding paper have been graciously loaned the author by Dr. Evans and Dr. Courville[1]. It is unfortunate that the general plan for these papers requires that the histological descriptions of the various conditions must appear separately from the case histories and gross descriptions. In order to minimize this disadvantage, the histology will be discussed as nearly as possible in the same order as that followed in the discussion of the gross findings by Evans and Courville. One exception to the above seems to be desirable, *viz.*, that the discussion of the infectious granulomata precede the descriptions of the new growths of the VIIIth Nerve.

Little need be added to what has been presented in the preceding paper on the traumatic lesions of the cerebellopontile angle. There is nothing specific in the microscopic anatomy of fractures in this region. Minute changes in the VIIIth Nerve and its ganglia following trauma have already been covered.

Only one of the vascular lesions of this region, that of aneurysm, deserves further attention. Aneurysm, especially of the vertebral arteries, appears to be of considerable importance. Moser (cited by Henschen[2]) reports three cases of aneurysm of the vertebral arteries, two of them in old persons, no age being recorded in his third case. Although the histology is not presented, one is led to suppose that these lesions are on the basis of arteriosclerotic changes. The majority of aneurysms of the vertebral arteries, no doubt, have as their predisposing causes arteriosclerosis or congenital anomalies, or perhaps both combined. Marked inequality in size of the vertebral arteries is a congenital lesion fairly frequently seen at the autopsy table. Such congenital lesions predispose to arteriosclerotic changes and to the formation of aneurysm. Syphilitic arteritis is, perhaps,

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responsible for a very small percentage of the aneurysms found in the region of the VIIIth Nerve. Vascular syphilis tends to produce an obliterative endarteritis with fibrous thickening of the walls, a condition which usually prevents formation of aneurysm.

DEGENERATIVE CHANGES OF THE VIIIth NERVE DUE TO POISONS.

The effect of large doses of quinin and salicylates in producing changes in the auditory apparatus are well known. If these drugs are continued for some time cinchonism or salicylism results. Both of these conditions are accompanied by ringing in the ears, a feeling of fullness in the head and temporary deafness. Occasionally the deafness becomes permanent.

Hart[8] cites eight cases of neuritis of the VIIIth Nerve resulting in dizzy spells or deafness, which he believes are caused by nicotine. All of these patients but one had chewed tobacco to excess for periods varying from 10 to 30 years. The patient who did not use tobacco in the above form smoked 20 cigarettes per day. No other etiologic agent common to these patients could be found. Hart states that all patients improved markedly following partial or complete abstinence from tobacco. This clinical study is suggestive that nicotine may be one cause of VIIIth Nerve neuritis, but scientific proof is difficult to obtain.

PYOGENIC INFECTIONS INVOLVING THE REGION OF VIIIth NERVE.

Acute infectious lesions of the cerebellopontile spaces present no characteristic histologic changes. It is true that purulent exudates tend to accumulate in larger amounts in these areas. Since these conditions usually terminate fatally within a few days the specific effect upon the VIIIth Nerve is not of much importance. Although brain abscesses may bulge into the angle and produce symptoms of angle syndrome it is not necessary or desirable to discuss the history of brain abscess at this time. It is very important to know, however, that infectious material from the middle ear may pass by way of the lymphatics in the sheath of the VIIIth Nerve to the brain, producing local or general meningitis or a brain abscess.

Occasionally infections of the middle ear and mastoid extend to the surrounding bony framework. Two cases of osteomyelitis of the petrous portion of the temporal bone have been seen in the pathological service at St. Vincent's Hospital during the past year. These cases are briefly presented because of their unusual interest.

Case 1. History: The patient was a white male student, age 25 years. The chief complaint was headache and dizziness. On May 19, 1930, he had coryza followed by earache, which lasted about two

weeks. On May 24 the right ear was opened and a large amount of pus was discharged and the ear continued to drain for three to four days. The patient had throbbing in the head at this time. After the ear stopped draining he had a compressed feeling in his head, and his hearing decreased. Headache was present at this time, but there was no mastoid tenderness and no swelling. A simple mastoidectomy was performed, June 6, by Dr. Gundrum. The patient's hearing improved immediately and was fairly good until four days before his death. His hearing then became worse, headache was severe and his gait staggering.

Examination: The neurological examination showed a well marked peripheral facial weakness and very poor hearing, both on the right. The remainder of the examination was largely negative.

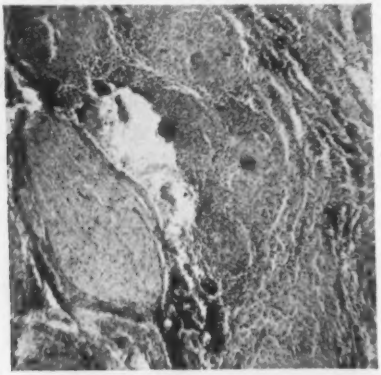

Fig. 1. Low power photomicrograph of meninges over the medulla in the case of Coccidiodal granuloma. Note the piece of cranial nerve to the left of the center surrounded by fibrous and granulation tissue containing a number of pseudotubercles and areas of heavy round-celled infiltration. The giant cell near the center of the field contains a large spore body.

The temperature varied from subnormal to 102° F. The urine showed no changes of importance. The blood examination revealed a hemoglobin of 65 (Sahli), 4,340,000 red blood cells, 10,800 white blood cells, with 50 per cent lymphocytes and 50 per cent polymorphonuclear leukocytes.

The patient was admitted to the hospital July 3, 1930, and died on July 6, 1930.

The autopsy was performed by Dr. B. O. Raulston.

Autopsy: There was a diffuse purulent meningitis with the purulent exudate somewhat more abundant at the base of the brain.

There was pus in the middle ear on the right side and in the labyrinth. The mastoid cells had been recently drained on the right. There were one or two carious places in the petrous bone from which pus was draining. On chipping away a portion of the bone with a chisel it was found to be a mere shell with irregular necroses ramifying throughout the bony process. These cavities were all filled with greenish pus. The necrosis had extended to the auditory canal and the VIIth and VIIIth Nerves were bathed in pus.

Case 2. History: The patient was a married man, age 35 years. The chief complaint was pain over the right side of the head. About July 1, 1930, the patient had a severe cold, which localized in the right ear. About a week later the drum ruptured and the ear discharged pus. The ear had been painful and had discharged more or less since the onset.

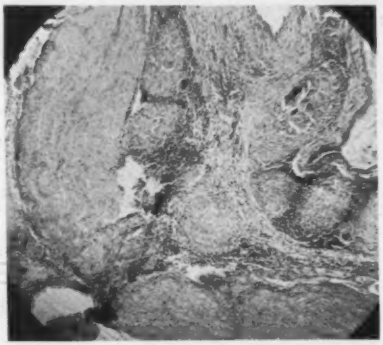

Fig. 2. Low power view one field removed from Fig. 1 showing along left side of field longitudinal section of one of cranial nerves embedded in Coccidiodal granulation tissue.

Examination: Temperature on admission to St. Vincent's Hospital was 90° F., pulse, 100; blood pressure, 140/74. There was a small amount of pus draining from the right ear.

The laboratory examination revealed the following: Hemoglobin, 59 (Sahli), 4,100,000 red blood cells, 13,000 white blood cells, and 75 per cent polymorphonuclear leukocytes. The urine was normal.

A mastoidectomy was performed on July 27, 1930, by Dr. J. M. Brown. The right mastoid was large, pneumatic and full of granulations, pus and diseased bone.

The patient did well until Aug. 2, 1930, when he had a severe chill. The temperature rose from 98.8° to 102.6° F. A few hours later

the patient became delirious, passed into coma and died three hours after the onset of the delirium.

Autopsy was performed by the author 3½ hours after death.

The skull cap showed rather deep depressions over the convolutions. There was a moderate quantity of thin purulent fluid in the pia-arachnoid over the convexity of the brain. A greater amount of greenish pus had accumulated about the base of the brain, being especially abundant under the tentorium along the brain stem. Considerable pus was present along the course of both Sylvian fissures. There was pus in the middle ear. On opening the labyrinth it also was filled with thick greenish pus. The petrous portion of the temporal bone showed marked necrosis. Several irregular carious spaces, each about a centimeter in diameter, connected by tortuous channels in the bone, were filled with pus. A lancet-shaped Gram positive diplococcus was isolated from the pus. The dura over the mastoid area was intact.

Comment: These two unusual cases occurred within a month of one another. They illustrate the uncommon complications of the middle ear and mastoid infections resulting in marked bony destruction in the region of the seventh and eighth nerves.

THE INFECTIOUS GRANULOMATA IN RELATION TO THE VIIIth NERVE.

1. *Tuberculosis:* Of the infectious granulomata, tuberculosis and syphilis are most often responsible for lesions of the cerebellopontile angle affecting the VIIIth Nerve. Six cases of tuberculoma of the cerebellum and brain stem simulating angle tumors in symptom-complex have been collected by Henschen[2] from six different authors.

A brief review of two of these cases will suffice to illustrate the effects of tuberculoma in this region.

The first case is one taken from a report by von Banze[4]. The patient was a boy, age 4½ years.

Clinically, there was headache, vomiting, a tendency to fall forward, left-sided abducens and facial paralysis. There was also difficulty of hearing on the left. Motor disturbances of the extremities existed on the right side.

The autopsy revealed a tubercle the size of a nut in the left corpus restiforme and just outside of this a second the size of a bean. On the left side the eighth to the tenth nerves were caught in the tumor. Moreover, seven other conglomerate tubercles were found.

A second case, taken from a report by Manasse[5], follows: The patient was age 43 years. On the left side the tympanic membrane was normal, while there was a well developed facial paralysis on the same side. The autopsy revealed in the posterior part of the left

side of the pons a tumor the size of a hazel nut. The acusticus and facial nerves were embedded fast in the tumor mass. A second tumor located near the first one was embedded in the cerebellum. A portion the size of a bean projected into the pia. The vagus group of nerves were fixed in this tumor. Other tubercles were seen in other parts of the brain.

The histology of tuberculoma is the same here as elsewhere in the brain. It may be noted, however, that tuberculomata have a tendency to become multiple and the symptom complex may be confused by the presence of tuberculous tumors in other parts of the brain, other than in the cerebellopontile angle.

Following tuberculous middle ear disease the petrous portion of the temporal bone may become the seat of a tuberculous infection.

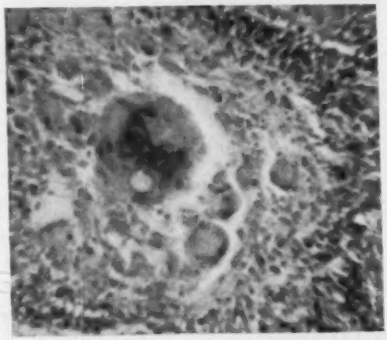

Fig. 3. High power photomicrograph of multinuclear giant cell containing a large doubly-contoured spore body. Same cell as seen in Fig. 1.

Hayden and Cohen[6] present a brief summary of such a case in a 14-year-old negro boy. There was a marked erosion and a moth-eaten appearance of the petrous portion of the temporal bone on the left involving about two-thirds of the bone. The bony cavities were filled with pus and caseous material. This is a case of tuberculous osteomyelitis of the temporal bone involving the whole VIIIth Nerve.

Tuberculous middle ear disease which goes on to an osteomyelitis of the petrous portion of the temporal bone, causing destruction of the bony framework about the VIIth and VIIIth Nerves, may progress about as follows, according to Hayden and Cohen: The disease in the middle ear may be acute or chronic, the latter is the more common. Tubercles form in the mucous membrane of the

middle ear. These become caseous and ulcerate, resulting in multiple perforations of the drum membrane. The perforations soon coalesce and destroy the drum completely. These authors state that painless perforation of the drum in the posterior superior quadrant is characteristic of tuberculosis of the middle ear. Following perforation of the drum, a scant watery, cloudy or bloody discharge comes from the ear. The discharge soon becomes profuse and purulent, due to secondary invaders. Pale, friable and edematous granulations tend to fill the space about the ossicles. Necrosis of the underlying bone results with the formation of large sequestra. The facial canal is attacked and facial paralysis results early. If malignancy can be excluded, facial paralysis occurring early in middle ear disease indicates tuberculosis. Milligan[7] states that facial paralysis occurred in 45 per cent of cases of proved tuberculosis of the middle ear, while it was found in only 2 to 5 per cent of nontuberculous middle ear disease.

Occasionally tuberculous meningitis becomes more or less chronic. The author has seen two such cases in which the meninges at the base of the brain had become extremely thick due to development of tuberculous granulation tissue. In one of these cases, that of a young Indian about 25 years of age, there had been definite symptoms of intracranial involvement for a period of one-and-one-half years. The autopsy revealed a thick, shaggy, semigelatinous granulation tissue of considerable thickness, especially over the base of the brain. In the cerebellopontile angles there was a network of thick gelatinous strands in which indefinite gray tubercles could be found on close inspection. The clinical men interested in the case were loath to accept the diagnosis of chronic tuberculous meningitis until acid-fast bacilli were demonstrated in the smears from the meninges. The histology was likewise characteristic of tuberculosis with many large tubercles present in the fibrous and tuberculous granulation tissue, showing caseous necrosis and Langhan's giant cells. There was an ulcerative tuberculosis of the right lung.

The second case consisted of a brain sent in to the Department of Pathology at Stanford University Medical School for diagnosis. The greatly thickened meninges were almost identical with those described above.

Although these cases are not illustrative of specific lesions localizing in the angle, they do represent interesting conditions which may affect several or all of the cranial nerves to a variable degree.

Coccidioidal granuloma of the meninges may occasionally produce tumor-like nodules resembling tuberculomata. The author has per-

formed an autopsy in such a case. A brief account of this case follows:

2. Coccidiodal Granuloma of Meninges Simulating Angle Tumor: The following case report is submitted as illustrating a rare condition which must be considered in connection with pathology of the VIIIth Nerve, especially in California.

History: The patient was an American, white male, age 25 years. At the time of his admission to Lane Hospital, San Francisco, he complained of attacks of dizziness, fainting spells and vomiting, which had begun two months previously. He had had frontal headaches, slight stiffness of the neck, loss of memory for the past few

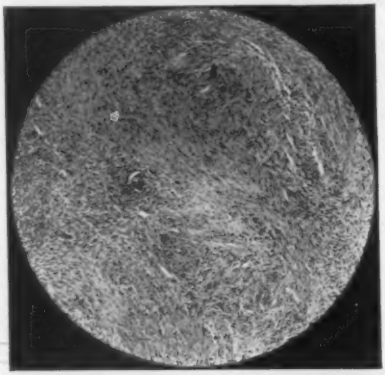

Fig. 4. Neurofibroma of the VIIIth Nerve (No. 1) showing the whorled arrangement of the connective tissue in the denser fibrous part of the tumor (low power), H. & E.

months and slowing up of speech and cerebration. He had not felt well for six months and had stopped work four months previously.

Examination showed some occipital tenderness, definite bilateral choked discs and slight irregular nystagmus. Examination of the cranial nerves showed no definite impairment of function except in the vestibular branch of the VIIIth Nerve.

Test, Nov. 6, 1926: Patient in sitting posture. *1.* Head tilted quickly A. L., falls to left, causes nausea. *2.* Head tilted P. R., nausea. *3.* Head tilted A. R., normal. *4.* Head tilted R. L., normal. Further tests impossible because of nausea.

Conclusion: Suggestive of trouble around left temporal with involvement of left anterior vertical semicircular canal.

The spinal fluid did not form a pelicle on standing. On the probability of finding an angle tumor, a cerebellar exploration through two

large windows in the occipital bone was made, Nov. 11, 1926. A right ventricular puncture produced 30 to 40 c.c. of clear fluid under pressure. In the region of the cisterna magna there was an opaque fibrofibrinous membrane covering the fourth ventricle. No tumor could be seen in the angle. A portion of this membrane removed for histological examination showed a number of giant cells and a small "tubercle."

The patient improved for a short time following operation, then gradually became worse, and died Jan. 24, 1927.

The clinical diagnosis following the exploration was tuberculoma of the brain.

Autopsy: The postmortem examination was limited to the head. There were two raised, yellowish nodular growths; one in the skin over the right eye, the other near the left sternoclavicular joint.

The cerebellum was considerably softened and firmly adherent to the dura which covered the windows in the occipital bone. There was marked fibrous thickening of the pia over the base of the brain, forming a grayish-white, opaque veil which extended from the optic chiasma back over the pons and medulla. Numerous small translucent nodules could be seen in the thickened pia. A yellowish caseous nodule, 1 x 0.4 c.m., was found on the under surface of the medulla; also a similar nodule on the right side in the cerebellopontile angle.

Microscopic examination of a section through the medulla shows an extreme fibrous thickening of the pia in which numerous cellular pseudotubercles occur (see Figs. 1 and 2). These contain exceptionally large Langhan's giant cells. Several of the giant cells contain within them large spherical sporelike bodies with doubly refractile capsules (see Fig. 3). One of the largest of these shows beginning formation of endospores. There is heavy round-cell infiltration of the connective tissue. Other areas show partly hyalinized fibrous tissue, areas of caseation and calcification. Smears from the meninges revealed no acid-fast bacilli and no other bacteria. Several large spherical sporelike bodies with doubly refractile walls were found.

Comment: This case adds another disease of the infectious granulomata to the group that may produce symptoms simulating angle tumor.

3. Syphilis of the Eighth Nerve: Lund (cited by Karl Grahe[8]) found neurolabyrinthine changes in 20 per cent of 515 cases of neurosyphilis personally observed by him. Kobrak (cited by Orleansky) reported that in 106 cases of untreated luetics only 19 were found with sound acoustic nerves. Orleansky says that syphilitic

infection of the inner ear is seen early in either the second or third stages of the disease or it may occur in congenital syphilis. The earliest cases have been seen six weeks after infection, but most often four to twelve weeks following the primary lesion. In congenital syphilis the involvement of the acoustic nerve may come at any time between the sixth and thirtieth years.

Beck[9] reported a case of gumma of the acoustic nerve with complete paralysis of the seventh and eighth nerves, and partial involvement of the third, fifth and ninth nerves. Antiluetic treatment restored, in part at least, the hearing on the affected side.

Henschen[2] in his monograph on tumors of the VIIIth Nerve devotes less than a page to a discussion of the luetic lesions. Luetic

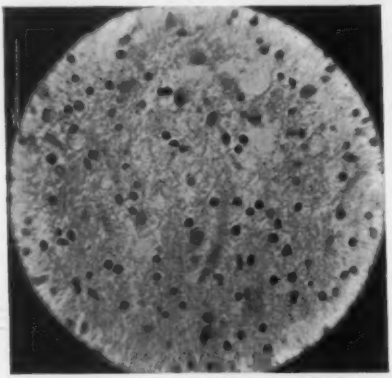

Fig. 5. The reticular portion of a neurofibroma (No. 1) of the VIIIth Nerve (high power), H. & E.

affections of the cerebellopontile angle are usually a part of a more or less diffuse gummatous meningitis, which in a large percentage of cases results in impairment to the VIIIth Nerve. Siemerling's[10] case, cited by Henschen, is an example of diffuse gummatous meningitis. In the beginning, according to Kaufmann[11], the gummatous tissue is spongy, colloid or richly cellular. The gummatous areas then undergo caseation and later become fibrous. The caseation may be diffuse or more often it occurs in spots. The caseous spots turn yellow, while the tissue between is gray-translucent and white fibrous. The gummatous tissue infiltrating the meninges produces firm adhesions, which are most abundant around the Sylvian fissures. Changes about the vessels are prominent and consist of periarteritis, mesar-

teritis and endarteritis. Fairly frequently the vessels may become obstructed. The gummatous overgrowth extends to the epineurium of the cranial nerves and infiltrates the adventitia of the blood vessels. The optic and oculomotor nerves are most frequently attacked (Kaufmann), resulting in thickening and atrophy. Any of the cranial nerves may be involved, however.

Microscopically the gray areas are composed of short, small spindle cells with groups of lymphocytes, plasma cells and eosinophiles. Numerous epithelioid cells are present, which appear granular and cloudy, with poorly stained nuclei in contrast to the epithelioid cells seen in tuberculosis. Occasional Langerhans' giant cells may be found bordering the caseous areas.

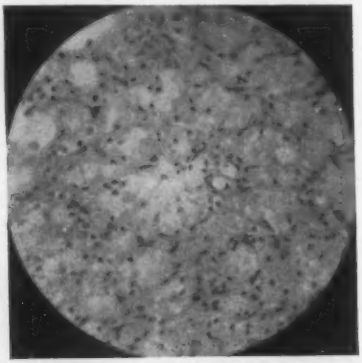

Fig. 6. One of the degenerating areas in a neurofibroma (B-2033) showing pale vacuolate areas due to microphages loaded with lipoid (low power), H. & E.

Besides the more diffuse process outlined above, gummatous tumors may localize in the cerebellopontine angle or in the surrounding structures, thus setting up a symptom-complex simulating that produced by a neoplasm.

The pathological changes in the labyrinth and in the auditory nerves in tabes are briefly discussed by O. Mayer[12], who has recently investigated four cases. In three cases he found very marked atrophy of the cochlear branch of the VIIIth Nerve, also atrophy of the ganglion cells of the spiral ganglion, thus incapacitating the organ of Corti and producing changes in the vestibular branch as well. In the fourth case he found marked changes in the organ of Corti characterized by otosclerotic foci. He found that the tabetic changes

in the VIIIth Nerve begin in the peripheral part of the nerve trunk. Mayer failed to give a detailed description of his microscopic findings.

Upenskaja presented a case (cited by Knick[13]) in which a more detailed histological study was made. He cites the case of a woman, age 57 years, who had had tabes for fifteen years, had been hard-of-hearing for one year, and nearly deaf for six months. Examination of the brain stem along the entire course of the acoustic fibres revealed the fact that the various nuclei were strongly affected. They were poor in cells, the ganglion cells were pycnotic, especially in all of the ventral nuclei, the tuberculum acusticum and, to a limited extent, in the angular nucleus. The most marked changes were in

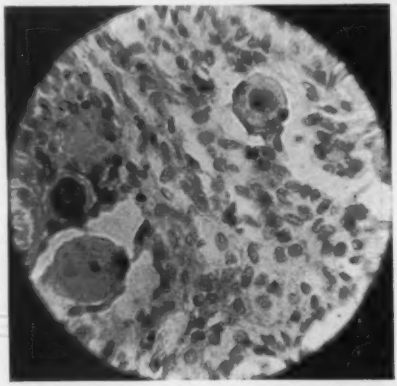

Fig. 7. Photomicrograph (high power) of a psammoma showing the characteristic type of cell composing the meningiomas. Several psammoma bodies with their concentric rings are shown. H. & E.

the cerebellopontile angle, where a convolution was in part atrophied. Proliferation of glia was found in the areas of degeneration. The vessels of the brain stem were all markedly changed, the walls thickened, and hyaline endarteritis was present. Many plasma cells and histiocytes were found in the adventitia.

The leptomeninges were thickened and infiltrated, especially along the vessels.

TUMORS OF THE VIIIth NERVE.

The histology of the VIIIth Nerve is discussed by Dr. Courville in the first paper[14] of this series. Suffice it to say here that the nerve trunk in its peripheral portion is made up of the same components as are found in other nerves, *viz:* axons and myelin sheaths

enclosed in their neurilemmas. These fibres are bound together by epineurium and the nerve trunk itself is covered by means of the perineurium. Henschen[15], in 1915, described two different components of the VIIIth Nerve, *viz:* glial and nonglial elements, corresponding with central and peripheral portions, respectively. It is necessary to understand this fact before the tumors of the VIIIth Nerve can be intelligently studied. The details in arrangement of the two tissues and their respective cell types have already been discussed in the first paper of this series.

There appears to be some question as to the origin of certain elements composing the acusticus tumors. The terms fibroma and neurifibroma indicate that the denser fusiform cells are considered to be

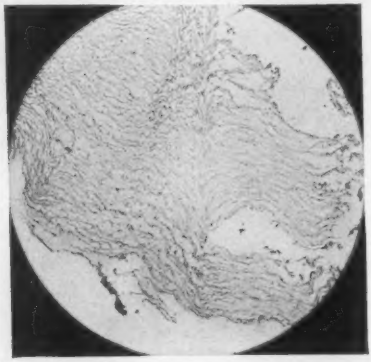

Fig. 8. Photomicrograph (low power) showing the delicate lace-like structure of the cholesteatoma. A few small round cells may be seen grouped here and there.

of connective tissue origin. Some investigators regard the smaller cells with oval nuclei, which occupy the less cellular or edematous areas, as homologous with the sheath cells of Schwann and consider these to be the type cells of the acusticus tumors. Borst[16] was probably the first to make this suggestion, which has recently been elaborated by Skinner[17]. In line with this thought it is necessary, then, to regard the abundant connective tissue growth as a tissue reaction responding to the stimulation produced by the growth of the type cells. Skinner also believes that the neurofibromata generally are composed of neurilemma cells. Del Rio Hortega[18] states that the oligodendroglia is absolutely homologous in the encephalomedullary centers with the Schwann cells of the nerves. Goodpasture[19],

on the other hand, thinks it unnecessary to conclude that the two types of cells seen in the neurofibromata of the VIIIth Nerve are genetically different. He suggests that the reticular areas are less differentiated parts of the tumor which may later progress to a more fibrous form.

Penfield[20], 1927, agrees with Mallory[21] in his classification of the acusticus tumors as perineurial fibroblastomata in contradistinction to the neurofibromata which develop in connection with the peripheral nerves. Penfield concludes that these tumors do not contain neuroglia nor do they contain nervous elements, but represent a particular form of collagen. He, therefore, confirms Mallory's idea that the fibroblast is the type cell of the nerve sheath tumor as well as

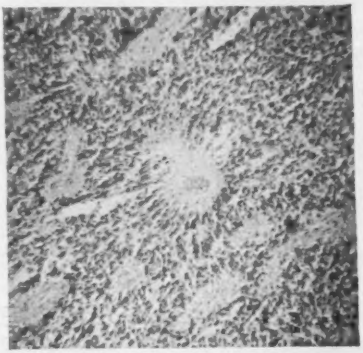

Fig. 9. Photomicrograph (low power) of the ependymoblastoma. The clear, myaline areas about the vessels are characteristic. H. & E.

of the so-called "dural endothelioma." The perineurial fibroblastoma must be considered to arise from the perineurial or endoneurial connective tissue which invests nerve bundles and fibres. The loose reticular areas found in the acusticus tumors are considered by Penfield to be due to degenerative changes.

Degenerative changes are found in many of the neurofibromata of the VIIIth Nerve. Perhaps the commonest one is that due to the accumulation of lipoid in large phagocytic cells, giving to the section an hydropic appearance (see Fig. 3). This material stains well with Scharlach R and appears to be largely cholesterol since it transmits light with crossed Nicol's prisms (Cushing[22]). It is found both in and outside of the cells. Pigmentation due to hemorrhage or diapedesis of red blood cells is quite frequently seen. There

is a marked tendency also to develop hyaline areas, especially about the blood vessels.

Alexander and Obersteiner[23] report finding small homogeneous bodies in the cochlear portion of the nervus acusticus near the junction of the glial and peripheral portions of the nerve. These bodies have the staining reactions of corpora amylacea. They seem to develop in the later periods of life and are attributed to the regressive changes in the astrocytes in the peripheral part of the glial portion of the nerve. The authors question the possibility of any clinical significance in relation to this phenomenon.

HISTOLOGY OF THE ACUSTICUS TUMORS.

A general description of the gross characteristics of the neurofibromata of the VIIIth Nerve without discussing any specific tumor has already appeared in the previous paper[1]. No attempt will be made here to enlarge upon this particular phase. To discuss the histology of a group of specialized neoplasms without the accompanying case histories or gross descriptions is like going to sea without compass or rudder and likely to lead nowhere. Furthermore, the excellent work on the acusticus tumors by Cushing[22] and others make the matter largely superfluous. For the sake of completeness, however, several neurofibromata are described in order to illustrate their various histological characters.

1. *Neurofibroma* (coroners' case): Histologically this tumor is composed of two kinds of tissue, *viz:* a dense cellular fibrous tissue and a loose reticular tissue with fewer cells. The two types are well intermixed. Whorls of fusiform cells of somewhat variable size, resembling in their arrangement the whorls of smooth muscle cells in a fibromyoma, are seen throughout the section (see Fig. 4). In between these denser areas are seen small, round or oval, dark-staining nuclei surrounded by irregular, poorly outlined cytoplasmic borders. In general, the cell bodies are roughly star-shaped and drawn out into fine fibrils which occupy the intercellular spaces (see Fig. 5). Occasional larger cells with poorly defined cytoplasm occur in these areas. The tumor is quite vascular, especially in the reticular areas. The vessel walls are relatively thick and hyalinized.

Mallory's aniline blue stain shows whorls of fairly dense fibrils which stain a dull purplish while the connective tissue about the vessels is bright blue. The reticular areas show a fine network of almost colorless fibrils. The cytoplasm stains poorly or not at all, while the nuclei stain orange red.

2. *Neurofibroma* (A-3046): This tumor presents two types of tissue, *viz:* the fibrous and reticular. In the small pieces available

for section the differentiation of the two types of tissue is less well defined than in the first case. Small palisades and whorls of fusiform cells are seen with the reticular areas quite intimately intermixed. Larger areas are found in which the tissue is largely reticular, but even here narrow strands of fusiform cells can be seen traversing these areas. Several hyaline areas are seen among the fibrous groups. This tumor is also quite vascular. In one section there is a considerable hemorrhage.

3. Neurofibroma (11-150): Sections of this tumor are remarkable for their great vascularity. There is a comparatively small amount of fibrous tissue with a correspondingly greater amount of cellular reticular tissue. Many large thin-walled vessels are present and con-

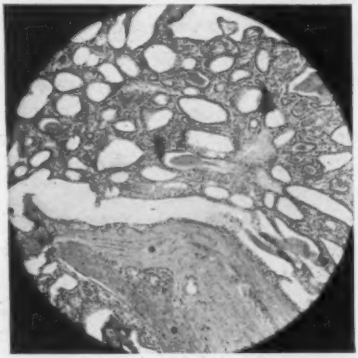

Fig. 10. Photomicrograph (low power) of the peculiar epithelial tumor of the cerebellopontile angle. Note the acini lined by high columnar epithelial cells. Many of the acini contain hyaline colloid-like material. H. & E.

siderable hemorrhage is evident. In this tumor the glial portion of the nerve has proliferated at the expense of the fibrous part. Grossly the tumor was softer than the usual neurofibroma and its growth had been comparatively rapid. This neoplasm, therefore, shows gliomatous tendencies and is potentially more malignant than the usual neurofibroma.

4. Neurofibroma (B-1401): There is nothing unusual about the histological characteristics of this acusticus tumor. Fibrous and reticular tissue are present in abundance; neither are especially predominant. In places the fusiform cells have produced considerable collagen. The tumor is only moderately cellular. Mallory's connective tissue stain shows a relatively heavy framework of purplish

fibrils. This tumor is just the opposite to the preceding one, in that it shows more of the characteristics of a fibroma and fewer of the gliomatous changes.

5. *Neurofibroma* (B-2033): Sections show whorls and palisades of fibrous tissue which are quite sharply defined from the reticular areas. The latter differ from those already described in the presence of many large phagocytic cells containing lipoid. These pale, finely granular cells give the reticular tissue an hydropic appearance (see Fig. 6).

6. *Neurofibroma* (5628): Microscopic examination of this tumor reveals a structure resembling superficially a glioma. The extreme vascularity strengthens this impression. The tissue appears very cellular (Van Gieson stain being composed of elongated cells with oval to fusiform nuclei of variable size). On the whole, this tumor contains more of the fibrous elements than do most of those described. The fibrous tissue is cellular and quite embryonic in type, presenting the picture of a fibrosarcoma. There are, however, small areas scattered throughout in which reticular tissue of a peculiar edematous or hydropic type can be found. In these areas many large clear spaces appear in the cytoplasm and even in some of the nuclei small, clear vacuoles may be made out.

Sharlach R stains reveal considerable amounts of lipoid. The lipoid occurs in large and small droplets, most abundant on either side of the nuclei of the fusiform cells.

Perdrau's stain shows abundant small, wavy fibrillae in the fibrous areas. Very few fibrils are present in the reticular areas. Many round, clear spaces are present in these areas.

MENINGIOMA (PSAMMOMA).

The particular tumor described by Evans and Courville[1] as Case 5 is composed largely of fusiform cells which are rather plump, supported by a connective tissue framework. The nuclei are oval or slightly elongated and only moderately rich in chromatin. In general, the growth is vascular and these cells are of the type that are usually described as endothelial. They tend to be arranged in whorls and often near the center of the whorl there is a hyaline pink-staining psammoma body (see Fig. 7). These are concentrically layered hyaline bodies containing calcium.

CHOLESTEATOMAS.

These tumors, which are said to originate from epithelial rests, occur occasionally in the cerebellopontile angle. A brief description of the histological characters of the tumor presented as Case 4 in

the preceding paper[1] follows: The structure is very simple, consisting of concentrically arranged wavy lines which take the aniline blue of Mallory's connective tissue stain. There are no tissue cells discernible, but here and there small groups of round cells appear. A good idea of the microscopic appearance may be had by referring to Fig. 8.

The tumors of the glioma group which may localize in the structures about the cerebellopontile angles are not described here since their histology is the same as that of the various gliomas elsewhere in the brain.

EPENDYMOBLASTOMA.

Following is the histological description of the ependymoblastoma of the fourth ventricle and the right cerebellopontile angle described by Evans and Courville as Case 6.

L. A. C. G. H., Case 137-750. Sections stained with hematoxylin and eosin show the characteristic clear areas surrounding the small vessels. The clear areas in turn are bordered by large round to oval nuclei arranged in one to several rows (see Fig. 9). The nuclei are large and filled with finely granular chromatin. One or two small eccentrically placed nucleoli are usually present. Between the clear spaces the tissue is very cellular. No mitoses are seen. Several small hemorrhages are present.

In the silver preparations fibrillary processes extending from the nuclei can be seen crossing the clear areas to the walls of the vessels.

Mallory's connective tissue stain shows the fibrous tissue confined pretty well to the walls of the vessels.

EPITHELIAL TUMOR.

L. A. C. G. H., Case 70-181. This remarkable tumor has a close resemblance microscopically to hyperplastic thyroid gland. There are large and smaller, more or less irregular acini, many of which are filled with a pink-staining homogeneous substance, resembling colloid. The lining cells are cuboidal to cylindrical and have deeply-staining, peripherally placed nuclei (see Fig. 10). The cytoplasm is extremely clear in many of the cells, making the resemblance to thyroid tissue even more exact. A definite fibrous framework can be seen in between the acini, accompanied by small groups of epithelial cells like those lining the acini. In places the tumor is very loosely constructed, due to the presence of large spaces, some of which appear to be vascular spaces, while others are lined by epithelium. Along one border of the tumor there is a considerable amount of dense hyaline

fibrous tissue in which there is a great deal of hemosiderin. In one of the broad fibrous bands there is an area of dense calcification.

Perdrau's silver stain shows an intricate network of black fibrils (connective tissue) between the acini. The hyaline material in the acini stains black also.

Mallory's connective tissue stain shows the dense fibrous tissue staining a deep blue, the more delicate framework composed of pale-blue fibrils. The colloid and the nuclei of the epithelial cells stain deep red, while the cytoplasm is somewhat paler.

Sharlach R haematoxylin stains show a large amount of lipoid present in the epithelial cells and in the interstitial tissue. The lipoid is quite irregularly distributed.

SUMMARY.

1. The effect of nicotine on the VIIIth Nerve is briefly discussed.

2. Two cases are presented of osteomyelitis of the petrous portion of the temporal bone following middle ear disease.

3. The infectious granulomata affecting the cerebellopontile angle are discussed. A case of coccidiodial granuloma producing granulomatous nodules localizing in the angle is also presented.

4. The histology is described of six different neurofibromata of the VIIIth Nerve.

5. Other tumors described histologically include a meningioma of the psammoma type, a cholesteatoma, an ependymoblastoma and an epithelial tumor of unusual type.

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3551 University Avenue.

Professor Erich Ruttin, of Vienna, is at present making a lecture tour of this country. He has given lecture courses in about a dozen large centers and those of us who have been fortunate enough to have taken his course have no hesitancy in heartily recommending the otolaryngologic fraternity to make every effort to attend a course by this famous otologist. He will be in this country until August and still has a few open dates for lecture courses. The management of THE LARYNGOSCOPE will be glad to forward to Professor Ruttin all mail addressed to him care of this office.

THE NERVUS ACUSTICUS.

V.—THE VIIIth NERVE: SYMPTOMATOLOGY FROM THE STANDPOINT OF NEUROLOGY.*

DR. SAMUEL D. INGHAM, Los Angeles.

The eighth pair of cranial nerves, comprising the auditory and vestibular divisions, constitute an important part of the peripheral nervous system and so fall within the field of neurology. Both divisions of the nerve are purely afferent in type, *i. e.*, transmit impulses which arise in their end-organs toward the central nervous system. As with all afferent nerves, their end-organs are highly specialized and selective, and normally respond only to the specific stimuli to which Nature has attuned them by the process of evolution.

It may be assumed that the character of nervous impulses is fundamentally the same over all nervous pathways, and each sensory nerve is specialized in function, for the reason that its end-organ is selective, and because the nerve itself is normally protected from irritation, excepting the stimuli which arise in the end-organs.

Under abnormal conditions an irritation at any point along its course or central connections may give rise to impulses which pass over the normal central pathways and result in sensations and reflex actions similar to those which result from stimuli arising in the end-organ. Thus encephalitis of the brain stem or an angle tumor may cause vertigo, nystagmus and tinnitus, and it is often difficult to determine whether the end-organ or the central pathways of the nerve are irritated when these symptoms are present.

The symptomatology resulting from disturbances of the VIIIth Nerve may be classified into those due to irritation of the nerve or its end-organ; those due to loss or impairment of function; psychic reactions of irritation; and attempts at compensatory adjustments, physiological and philosophical.

Symptomatology Referable to the Auditory Division: Irritation of the end-organ or along the central course of the auditory nerve causes tinnitus. The cases with cochlear involvement belong to otology and are usually diagnosed by otologic tests. Those with intracranial lesions interest the neurologist and usually constitute only one detail in a picture of intracranial pathology. Loss or impairment of hearing is of importance neurologically not only as a localizing symptom of intracranial lesions, but also, in cases of chronic deafness, on account of the limitations it imposes upon the subject and the psychic reactions and personality changes that result. Deaf children, undiagnosed, are not only handicapped by their infirmity but

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are at times stigmatized as feeble-minded. In adult life the deaf person is not only limited in his contacts with life, but he is apt to be sensitive, seclusive, suspicious and emotionally unstable. Hallucinations of hearing are not uncommon in the deaf.

Symptoms referable to the disturbances of the vestibular nerve and of its intracranial connections are of great importance because of the intimate functional relationship which exists between this nerve and other important nervous mechanisms. This nerve is unique, in that through it various reflexes can be elicited by measured stimuli, the results having not only qualitative but also quantitative value. To spontaneous symptoms can be added the results of standardized tests which are of great importance in diagnosis.

The symptoms of irritations of the vestibular nerve constitute a familiar and striking picture: vertigo; a special type of nystagmus; unstable equilibrium; forced movements; alterations in muscle tone and bodily attitudes. Prolonged irritation causes more or less prolonged disturbances in the field of the vegetative nervous system; pallor, sweat, nausea and vomiting, and a general feeling of collapse.

Symptoms of irritation of vestibular nerve may occur as a result of disease, but they may also result from unusual stimuli to normal structures, as in seasickness and neuro-otologic testings. The psychic reactions of the patient which follow turning chair tests and douching the ear with cold water sometimes makes it difficult to persuade him to return for further examinations.

Acute destructive lesions which result in permanent impairment of vestibular functions give rise to acute reactions in the early phase, followed by compensatory adjustments later.

Complete absence of all vestibular reactions to turning and douching, and presumably total absence of vestibular function bilaterally, is not incompatible with very satisfactory adjustments through vision and muscle sense. A wrestler with absence of vestibular function would not suffer from the "airplane spin." A college athlete with such a defect could throw the hammer successfully but could not keep his equilibrium running after a high "fly" in a baseball game.

The symptoms due to irritation of the vestibular nerve may be simulated by other conditions. This "dizziness" complained of by the patient is frequently not vestibular vertigo; jerking nystagmoid movements of the eyes should not be confused with systematized vestibular nystagmus; forced movements and disturbances of equilibrium are not limited to those of vestibular lesions; while pallor, sweat, nausea and vomiting may result from many causes. A careful observation of spontaneous symptoms followed by the application of standardized tests of the vestibular reactions makes possible a

reasonably accurate evaluation of the functional capacity of the vestibular apparatus.

Intracranial lesions not directly involving the VIIIth Nerve may give rise to symptoms of disturbance of the auditory or vestibular functions.

Deafness may be due to bilateral lesions of the cortical areas in the first temporal convolutions. Unilateral lesions in these areas, however, are almost symptomless, the usually assumed explanation being that both auditory nerves are in functional relationship with both cortical auditory areas, much as both optic nerves are in functional relationship with both cortical visual areas. It is a curious fact that brain stem lesions above the auditory nuclei very rarely cause noticeable impairment of hearing. Lesions of the temporal lobe (second temporal convolution) in the left hemisphere cause word deafness which, when of severe degree, may give the impression of deafness.

Since stimuli from the vestibular end-organ result in conscious perceptions of definite movements as in the rotation tests; and since false impressions of movements (vertigo) occur when convection currents are produced in the semicircular canals by caloric stimulation, it seems reasonable to assume that the cortical cerebral area exists in functional relation with the vestibular nerve. No such area has been actually demonstrated, but Dr. Mills has postulated the existence of such an area in the temporal lobe. Irritations of this area might explain some types of dizziness, and destructive lesions at the same location would result in impairment of some of the vestibular functions.

Symptoms referable to disturbances of the eighth cranial nerves and their central connections are frequently associated with other symptoms, due to the involvement of neighboring structures; to increased intracranial pressure; and to infections, especially syphilis, meningitis and the exanthemata.

The cerebellopontile angle tumor is a classic example of intracranial lesion involving the VIIIth Nerve, giving rise to symptoms of irritation and destruction of both the vestibular and auditory divisions of the nerve, associated symptoms of involvement of the brain stem and cerebellum, and evidences of increased intracranial pressure. Psychic reactions of the patient to the entire situation should also be added to the picture.

From the standpoint of neurology, VIIIth Nerve symptoms are analyzed and evaluated in their relation to the history of the case and to associated symptoms and findings from examination, in an effort to draw a recognizable clinical picture and so ultimately to establish a diagnosis.

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THE NERVUS ACUSTICUS.
VI.—VESTIBULAR SYMPTOMATOLOGY.*

DR. EUGENE R. LEWIS, Los Angeles.

On the program I am assigned, "Symptomatology of the VIIIth Nerve-Vestibular." A student who was called upon to discuss the lions on the Peloponnesus answered, "There were no lions on the Peloponnesus," and sat down. Vestibular service, like all physiologic services, is without "symptoms" under normal service conditions. It is only when something disturbs working conditions sufficiently that symptoms are noted. Instead of following the student's example by resuming my seat at this point in the series of talks on the VIIIth Nerve (the last of the *vestibular* discussions) I am going to indulge in recapitulation and some generalizations.

The evolution of the human body has involved countless adaptations between the body and its environmental conditions. Among other entities to which the organism must adapt is *energy*, in many forms; among which are heat, light, sound and motion. When certain forms of organized life first broke away from their (hitherto) fixed natural habitat (hitherto fixed forms of life, such as corals, for example) and came to be tossed about by waves, *motion* entered for the first time into their *living conditions*. This new item of *motion* introduced necessities for new adaptations, and one of the mechanisms which evolved as a result developed into the vestibular apparatus, whose semicircular canals, maculae, end-organs, nerve paths and central and peripheral nerve and sympathetic connections we are studying. Other mechanisms subserving the sensing of gravity, posture, position, touch, sound, odor and light are also concerned more or less intimately with adaptations to motion. It is only necessary to contemplate the differences in organismal efficiencies respecting motion, as represented by lower forms, moved about by random wave motions and higher forms looping-the-loop in an aeroplane, in order to realize what evolutionary adaptation to motion really means. This *gradus ad Parnassum* may be actually traced in living organisms today, from the rudimentary end-organ cell in an otic pit, containing sea water and sand from the ocean, up through the conversion of this pit into a closed utricle, the rudimentary single semicircular canal offshoot from the utricle, sand replaced by otolith, sea water by endolymph, to the final elaborate "latest models" of invertebrate and vertebrate end-organ emplacements. Essential as are these intricate elaborations of the end-organ emplacements, they are only the *housings* of the *peripheral endings* of neuronal systems, whose operations represent the actual physiologic adaptation service

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of the vestibular apparatus. In these physiologic operations, the vestibular apparatus commandeers at all times the necessary co-operations of brain, spinal cord, sympathetic system, eyes, muscles, bones and innumerable other components of the body. (More accurately stated, all these *comprise the apparatus* which renders vestibular service). All of these evolutions concern only a *portion* of the means of adaptation of the organism to motion. The locomotive abilities of the deaf-mute lacking ear (vestibular) function demonstrates how well the organism can manage to get along without the internal ear part of this service. True, he may be seriously hampered by darkness (expert deaf-mute swimmers have drowned when the light has gone out, simply because they were unable to *see* where the surface of the water lay), but given visual service, he is able to walk, run or jump very much as a normal person does.

The vestibular physiologic mechanism which takes part in adaptative reactions of the body to motion is actuated by afferent nerve impulses originating in the internal ear. The ciliated end-organ cell is the initiator of these afferent impulses. Last week, Dr. Leon Myers demonstrated by model the peculiar pattern of arrangement of these end-organ cell groups—in three pairs of ampullae, *oppositely emplaced*—each pair at *opposite ends* of a pair of canals whose plane is at right angles to the planes of the *other two canal pairs*—representative of the three cardinal planes of space. By virtue of this peculiarly patterned disposition of end-organ cell groups, head movements of all kinds produce equal and opposite cilia disturbance of *functionally* conjugate ampullae. For example, the two horizontal canals lie in the same plane of the head, each ampulla at the *distal* end of its canal; *i. e.,* they are *"opposed."* Rotating the upright head to the right causes the cilia of the right crista to *trend toward* the right utricle, the cilia of the left crista to *trend away from* the left utricle, and these cilia disturbances of right and left ampullae are *simultaneous* and *equal* in degree. The same is true of *any conjugate pair* of *ampullae,* or of any possible combination of all pairs of ampullae.

Stimulation: Causing the cilia of a single ampulla to trend *toward* the utricle results in a discharge of afferent impulses of a certain sense and of certain intensity; causing these identical cilia to trend *away* from the utricle, results in a discharge of afferent impulses of opposite sense. For example, caloric stimulation of a definite amount (causing convection upward) in the anterior superior canal produces a sensation of vertical movement obliquely downward, lasting approximately 12 seconds; the *same amount* of reverse caloric stimulation of the same canal (causing convection downward) produces a sensation of vertical movement obliquely upward lasting approxi-

mately 6 seconds. The same difference in sense and intensity can be demonstrated respecting the horizontal canal hair cells. As the right and left canals are conjugated physiologically, the total effect of motion comprises the impulses coming from the major emission of the *right* crista and the minor emission of its *left* fellow, or *vice versa,* in all possible combinations of motion stimulus or in all head positions. For example, motion stimulus equaling the above mentioned caloric stimulation in intensity, would effect emission of impulses, the intensity of which differs in the two sides in the proportion of approximately 12:6; and the total impulse values reaching the brain would approximate 12+6=18. These numerical values are used for purely graphic purposes and are in no way to be understood as concrete. It is definitely known that a decided difference of this kind exists, and it is important to bear this in mind whenever reckoning with vestibular reactions.

Responses: The "reactions" observed in vestibular testing include vertigo, nystagmus, falling, past-pointing, pallor, sweat, nausea, vomiting and faintness. No discussion of these well known reactions is necessary, but it is well to note that the *emission of an impulse from the end-organs* results in *impulse disseminations* over a variety of nerve pathways, *whose nature,* extent and "patterns" must be clearly understood by examiners.

Qualitative: 1. The afferent impulse causes a *sensation* of motion; the sensory pathway is assumed to be the VIIIth nerve, medulla, inferior peduncle, cerebellum, superior peduncle, crus to cerebral cortex in the neighborhood of second temporal convolution of opposite side, some impulses reaching this cortical area of the same side. This pathway is presumably "normal" if stimulation causes a certain definite vertigo; but if it is *not* the kind normally resulting from the particular stimulation which has been applied—if, for example, after rotation to the right, vertigo *to the right* is produced, there is definite evidence of something abnormal. Abnormality is likewise apparent, if vertigo is experienced *without stimulation* or if stimulation produces *no vertigo.*

2. The afferent impulse results in certain eye *movements;* this pathway is assumed to be the VIIIth nerve, medulla, posterior longitudinal bundle, oculomotor-brain and ocular neuromuscular mechanism. This pathway is presumed to be "normal" if stimulation causes certain definite nystagmic movements; but if they are *not* the kind normally resulting from the particular stimulation which has been applied—if, for instance, with head upright, turning to right produces slow pull of eyes to left and quick jerk to right, there is definite evidence of something abnormal; likewise if the plane of eye

movement is off the horizontal, or if nystagmus occurs without stimulation, or if stimulation fails to produce eye movement.

3. The afferent impulse results in certain *fall* tendencies and *past-pointings.* This pathway is assumed to be the same as the sensory with the addition of the pre-Rolandic motor cortex, the internal capsule, and the pyramidal tract distribution. This pathway is presumed to be "normal" if certain definite stimulation produces *fall-tendency* and *past-pointing* of a certain definite kind; for example, after turning to the right, head upright, *fall-tendency* and *past-pointing* to the left in the horizontal transverse plane of the body. Just as in the other reactions already mentioned, if any other result is noted, it is evidence of abnormality.

4. The afferent impulse results in pallor, sweat, nausea, vomiting and faintness. This pathway is assumed to be the same as the sensory with the addition of the sympathetic and parasympathetic distributions. The vagus is included in this distribution, and this is sometimes spoken of as the "vagus" distribution; actually, it includes much more than the vagus connections.

This pathway is presumed to be normal if stimulation produces these results, approximately in the order named. If they fail to occur, or if they occur without stimulation, or if any other marked difference from normality be noted, it means something definitely abnormal.

Quantitative: There is a commensurate relation between intensity of stimulation and intensity of any of these reactions; thus quantitative considerations form an additional basis for detecting abnormality. For example, the ordinary person reaches the border line of his toleration of testing stimulation after approximately ten turnings to the right and ten turnings to the left, at the rate of one turn in two seconds; in a certain number of cases, somewhat more turning may be done, without inducing an undesirable degree of pallor, sweat and nausea. If, however, further turnings and caloric tests are made without producing discomfort or nausea, it constitutes definite evidence of abnormality.

Abnormalities: These abnormalities are of widely different natures, ranging from end-organ lesions, or lesions at one or more points along the pathway, to lesion of the cerebral hemisphere or cortex; also the lesion itself may be one of several of widely differing characters, ranging from *local* or *remote irritation* or *blocking,* of varying degree and recoverability, to irrecoverable *destruction;* and from single to multiple lesions, each of these again irritative, blocking or destructive, as the case may be.

In the interests of simplicity tonogenesis will not be discussed in this recapitulation, except to mention that the vestibular afferent

impulses, in common with other afferent impulses, take part in tonogenesis. Twenty years ago, Shambaugh stated that "the chief function of the vestibular apparatus is the emission of tonus impulses to the skeletal muscles." At that time I took issue with the statement, and I have had no reason to think differently since.

The end-organs of the maculae sacculi and utriculi participate in the emission of impulses along the vestibular nerve paths. Unanimity concerning their modus operandi is lacking; they seem specially concerned with reactions to position and posture, and probably participate also in all reactive adjustments to motion.

Earlier in this talk I mentioned that under certain conditions, fairly satisfactory adjustments to motion are evident in persons entirely lacking vestibular function. Lip-reading may compensate effectively for lack of hearing; vision may compensate effectively for lack of motion-sensing service ordinarily rendered by the ear; *but not in the dark*. In the fundamental neuromuscular patterns determining bodily adjustments to motion there are *three sources* of the *main determinant nerve impulses: vestibular end-organs, visual end-organs* and *deep sensibility;* additional impulses enter into the picture from tactile end-organs, smell end-organs and hearing end-organs, but these are of lower order, introducing additional refinements of stereognostic character. The efficiency of this adjustment mechanism in maintaining body relation with respect to the pull of gravity and the position of the body and its posture is obviously commensurate to its *intactness*. Without its service gravity alone would terminate survival at the first pitfall. At first blush one might be inclined to doubt the need for *providing against living upside down*. But Nature has developed a physiologic mechanism which actually provides against this contingency. The skeleton, skeletal musculature, viscera and all other integral portions of the body have evolved their forms and positional relations in the body on the basis of a fundamental positional and postural relation to the earth; feet down, head up; furthermore, the physiologic function of locomotion has likewise evolved upon the further basic detail of progression with the *eyes forward*. The proof of the necessity of providing against such contingency was shown in Vienna many years ago. Kreidl removed the sand from a crab's vestibular apparatus, substituting iron filings. The crab was then placed in his normal environment, with a magnet suspended above him. The *pull of gravity* upon the iron filings which served the crab as otoliths was reversed by the pull of magnetism, whereupon the creature reversed his positional relation respecting the earth and proceeded to rest and move about upside down exactly as it previously had rested and moved about right side up. This crab's behavior sheds light significantly upon the part played by

gravity in orientation and locomotion. Another important influence upon body reactions in orientation and locomotion is the force of inertia as sensed by "deep sense" and surface tactile sense; another influence is light, as sensed in visual impressions. The integrating action of the vestibular apparatus in sensing qualitatively and quantitatively each motion, large and small, to which the body is subjected is undoubtedly a far greater factor in orientations and locomotions than is realized. The fundamental neuromuscular "pattern" determining these body adjustments may be regarded as composed of *main outlines* with *detail shadings;* the *main outlines* of the pattern limned by vestibular, visual and deep sensibility impulses, the *shadings* by impulses coming from end-organs of smell, of hearing and of tactile sense. Erasing the shadings still leaves the pattern-meaning sharp and clear; erasing the lines impressed upon the pattern by either vestibular or visual impulses only, destroys very largely the utility and meaning of the pattern; for practical purposes one may consider that two of these three main impressions upon this "pattern" of service may suffice for ordinary orientation and locomotion needs, but *vestibular* alone, or *visual* alone, or *deep sense* alone do not suffice. The nature and extent of this inadequacy of neuromuscular mechanismal control may be realized by observing *vestibular* deaf-mutes in salt water, whose levitation counterbalances the pull of gravity on the body mass; or by observing blind persons under the same conditions of brine levitation. The deaf-mute will be fairly capable of controlling himself as long as he can *see* his positional relations; when deprived of visual guidance, he is practically helpless. The blind are surprisingly helpless when immersed in brine.

Upon a general conception of this kind of mechanismal control of the body one may build up the ability to realize the meanings of vestibular reactions and to reach certain conclusions of diagnostic import and value. Spontaneous phenomena observed in patients, and reactions to turning and douching, carefully observed and studiously considered in conjunction with the known fundamental facts, often reveal the location of a lesion as definitely peripheral, subtentorial or supratentorial. Experience shows that many cases present confusing findings which are inconsistent with the purely theoretical conception of what other more or less mandatory findings seem to indicate. Under such circumstances one is compelled to disregard a confusing inconsistency and be guided to diagnosis by the consistent remaining findings. Diagnosis based upon such careful analyses of sufficiently verified findings may be relied upon confidently, especially when supported by collateral observations from the history, general physical and laboratory findings.

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THE NERVUS ACUSTICUS.

VII.—EIGHTH NERVE SYMPTOMATOLOGY FROM THE STANDPOINT OF NEUROSURGERY.*

Dr. Carl W. Rand, Los Angeles.

The symptoms of acoustic nerve tumors as well as their pathology have been given so well by other essayists that I will not review them again. However, I will take the liberty of reading in some detail the history of a case which illustrates the progression of symptoms.

The patient, P. H., a male, age 46 years, mattress maker by occupation, came under observation in October of 1926. He complained of having had headache for some ten years, tinnitus and deafness in the left ear for one year, the latter symptom increasing. He also complained of double vision of about six months' duration, attacks of vertigo, unsteadiness of gait and occasional pain in the left side of the face. At times this pain was very severe and he was referred, not with the thought of his having a possible tumor in the cerebellopontile angle, but because of his facial neuralgia. Irritative symptoms of the fifth nerve or nucleus, while not common, may be present in cases of acoustic nerve tumors. The patient at this time presented such a symptom to a marked degree.

From the time he was thirty until 40 years of age he had occasional headaches, which at times were very severe and lasted four or five days. As a rule they would not last over one day, when he would go home, vomit, go to bed and the next morning wake up alright. These headaches would come at varying intervals of several days to a few months. During the last year the headache had been less prominent than formerly. A year prior to my first examination he noticed a ringing in his left ear. He thought he could hear the rumbling of street cars. This was almost constant for six months, being especially noticeable at night. It had gradually become less marked and at the time of my first examination had practically disappeared. During the previous year there had been impairment of hearing on the left side, which had increased until a point of almost total deafness was reached. For a period of six months before coming he had noticed double vision if he looked to the left. He also complained of dizziness and unsteadiness on his feet, especially if

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he had been sitting for a long time and got up suddenly. He tended to deviate toward the left. About four months previously he had a severe attack of pain in the left side of the face. It was described as sharp and shooting, it came on suddenly and was located in the lower jaw. It was so bad that he was unable to talk or eat. The attack lasted for about a week. Acoustic nerve tumors have been mistaken for trigeminal neuralgia on occasions because of the severity of the fifth nerve pain. Weisenburg[1] has described a case where a Gasserian operation was mistakenly carried out for relief of trigeminal pain caused by an acoustic nerve tumor which compressed the fifth nerve.

On examination he showed choking of both discs, which was of long standing, a marked spontaneous horizontal nystagmus on looking both to the right and left, and a vertical nystagmus on looking up. When looking to the left, he saw double. There was anesthesia of the left cornea. Hearing on the left side was practically gone for air conduction, but bone conduction was still present. The other cranial nerves were normal. Otoneurological tests revealed an absence of response from both vertical and horizontal canals on the left, and absence of response from the vertical canals, but presence of response from the horizontal canal, on the right. Roentgenological examination of the skull in Towne's[2] position failed to reveal erosion of the petrous ridge of the temporal bone. An operation was urged at the time but patient refused. He was indirectly heard from occasionally during the next four years. At the end of that time he was completely blind and there was complete nerve deafness on the left. He had developed convulsions, which were of the extensor or cerebellar type, and his ataxia had increased to such an extent that he could hardly walk. Headaches had again returned and were very excruciating, being accompanied by nausea and vomiting. At times he was incontinent. His sense of smell was entirely gone, probably as a result of high intracranial pressure. Choking of the discs had increased from 3 diopters in 1926 to about 6 diopters in 1930. The nystagmus was still present. The pain in the left side of his face no longer troubled him, but on the contrary an anesthesia of the trigeminal distribution in the left side of his face had appeared. He had developed difficulty in swallowing and his articulation was poor. His speech was of the type generally referred to as "bulbar speech." The patient was now on my service in the Los Angeles General Hospital, bedridden, and it was decided to attempt a ventriculography. Dr. Leo J. Adelstein had just started to do this under local anesthesia when the patient developed one of his cerebellar fits,

stopped breathing, became cyanotic and it was several minutes before he could be revived. About one month later, because of his terrific headache, I attempted to carry out a suboccipital exploration but again the patient developed a cerebellar attack and it was necessary to desist. After two months, Dr. George H. Patterson reopened the wound and succeeded in removing part of the floor of the skull, when he in turn had to stop because of another cerebellar episode. A short time later, he was again operated, when an encapsulated neuroma of the VIIIth Nerve was found. The capsule was opened and the contents scooped out as well as could be. This tumor was about as large as a golf ball. The patient subsequently died and the specimen has been presented to you earlier in the course by Dr. Newton G. Evans.

The accepted surgical approach to these tumors is by cerebellar exploration. The operative details will not be discussed, except to state briefly that usually a crossbow incision, as described by Cushing[3], is made in such a manner that two flaps can be turned back. Sometimes a straight midline incision is employed, as suggested by Towne[4] and Frazier[5], while again a curvilinear or horseshoe-shaped incision is preferred by others. The floor of the posterior fossa, together with the posterior half of the foramen magnum, are removed. The lobe of the cerebellum on the affected side is then elevated until the tumor is exposed. Again, part of the cerebellar lobe itself may be removed and the tumor "uncapped." This can be done without producing much ataxia, provided the dentate and other deep cerebellar nuclei are not injured. This procedure gives a better view of the tumor than elevation of the cerebellar lobe. Naffziger[6] has described an occipital flap, going above the tentorium and approaching the tumor through the tentorium. It is seldom used although it may be done as a second stage procedure after suboccipital exploration has already proved inadequate. An encapsulated pocket of fluid usually surrounds the tumor and in approaching it posteriorly the operator first comes down on this semitransparent membrane, which gives a bluish cast. After opening this and allowing the fluid to escape, one comes down on the true capsule of the tumor. This is easily opened and the contents of the tumor removed piecemeal, either with a blunt curette or an electric cautery. On occasion the entire capsule can be removed after the contents of the tumor have been taken away. In such a case a paralysis of the face on the side in question almost invariably results. Dandy[7] advised total removal of the tumor, to be followed later by an anastomosis of the paralyzed seventh nerve with either the spinal-accessory or

hypoglossal. There is still considerable discussion as to whether one should attempt a partial or complete removal of these tumors. I think it depends on the conditions found at operation. Even with incomplete removal of the tumor the patient in the majority of cases will have marked improvement over a period of years and, if necessary, a second operation may be carried out. It is needless to say that lumbar puncture, if carried out, should be performed with great caution as there is danger of herniation of the medulla into the foramen magnum. This is true of any posterior fossa tumor. Lumbar puncture, however, may be of some diagnostic value. Cases have been reported by Gardner[8] where compression of the jugular vein on the side of the tumor has shown no rise of the cerebrospinal fluid in the manometer, while compression of the opposite jugular vein will cause a distinct rise of this fluid in the manometer. In other words, there may be a spinal fluid block on the side of the tumor due to obliteration of the corresponding lateral sinus. With the abundant evidence, however, which can be elicited by neurological examination and neuro-otological and Roentgenological studies, lumbar puncture in cases of suspected acoustic nerve tumors is hardly to be recommended.

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BOOK REVIEWS.

Geschichte des Taubstummenproblem Bis ins 17 Jahrhundert. By Dr. med. et phil. Hans Werner, Ohrenarzt in Zurich. 276 pp., with 15 illustrations. Jena: Verlag Gustav Fischer, 1932. Price $3.50.

This volume of 276 pages is an unusually interesting presentation of the history and development of the problems of deafness and the education of the deaf from the earliest periods to the Seventeenth Century. In its six chapters it includes the medical literature, methods of instruction, social and intellectual status of the deaf, the pioneers in this field and their special contributions; the origin of the various systems of instruction and much additional interesting data, making a volume of fascinating and readable character replete with authentic information. It should be of special interest to all otologists and teachers of the deaf. M. A. G.

Das Gehorleidende Kind. By Karl Brauckmann, Leiter und Inhat der Lehr und Erziehungsanstalt fur Gehorleidende Kinder in Jena. 160 pages. Jena: Verlag von Gustav Fischer, 1931. Price $1.80.

In the preface to this monograph the author subdivides his subject-matter into three sections: (1) Speech Instruction to Congenitally Deaf Children; (2) The Conservation of Speech in Children Who Have Acquired Deafness; (3) Provision for the Instruction of Hard-of-Hearing Children.

In the discussion of these three important phases of juvenile deafness, Brauckmann renders a real service and contributes much of his personal experiences in this field that should be of distinct interest to all who may share in the responsibilities of this form of handicap in children.

Many pedagogic and scientific principles receive practical application in this little volume. It includes the evolution of speech, the significance of the nervous system, rhythm, accent, the psychological status of the deaf child and its gradual adjustment to social conditions; practical suggestions on voice and its functions, phonation, co-ordination, association, lip-reading, and a sketch of the development of this important phase of child training. M. A. G.

The Modern Conception of Deafness. By Harold Hays, M.A., M.D., F.A.C.S., Fellow, American Laryngological, Rhinological and Otological Society; Fellow, American Academy of Ophthalmology and Otolaryngology; Fellow, New York Academy of Medicine; former President of the American Federation of Organizations for the Hard-of-Hearing and of the New York League for the Hard-of-Hearing. St. Louis: THE LARYNGOSCOPE, 1932. 149 pages. Price $2.00.

This small, handy volume is a scientific treatise on the subject of deafness, written so simply that it can be understood by the layman as well as the physician.

This monograph has discussed this subject comprehensively. Not only are the new conceptions of anatomy and physiology dealt with, but attention is also given to the causation, symptoms and treatment of deafness, and special chapters are devoted to the social and economic problems of lip-reading.
interested in the deaf or hard-of-hearing.

This book should be in the hands of every otologist, physician and layman interested in the deaf or hard-of-hearing.